**Glenn Springs Holdings, Inc.**

# Baseline Human Health Risk Assessment

Newark Bay Study Area

October 2019

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**Acronyms and Abbreviations**

| | |
|---|---|
| ADAF | age-dependent adjustment factor |
| AOC | Administrative Order on Consent |
| ATSDR | Agency for Toxic Substances and Disease Registry |
| BaP | benzo(a)pyrene |
| BHHRA | Baseline Human Health Risk Assessment |
| BMD | benchmark dose |
| BMDL | BMD lower confidence level |
| CalEPA | California Environmental Protection Agency |
| CDC | U.S. Centers for Disease Control and Prevention |
| CERCLA | Comprehensive Environmental Response, Compensation, and Liability Act |
| COC | chemical of concern |
| COPC | chemical of potential concern |
| CSF | cancer slope factor |
| CSO | combined sewer overflow |
| CTE | central tendency exposure |
| DAF | dermal absorption fraction |
| DL | dioxin-like |
| DLC | dioxin-like compounds |
| DMA | dimethylarsinic acid |
| ED | exposure duration |
| ELCR | excess lifetime cancer risk |
| EPC | exposure-point concentration |
| FDA | U.S. Food and Drug Administration |
| FI | fraction ingested |
| GROS | Gamma Regression on Order Statistics |
| GSH | Glenn Springs Holdings, Inc. |
| HEAST | Health Effects Assessment Summary Tables |
| HED | human equivalent dose |
| HHCSM | human health conceptual site model |
| HI | hazard index |

| | |
|---|---|
| HQ | hazard quotient |
| IARC | International Agency for Research on Cancer |
| IRIS | Integrated Risk Information System (USEPA) |
| KM | Kaplan-Meier (calculator) |
| LADD | lifetime average daily dose |
| LMS | Linearized multistage |
| LOAEL | lowest-observed-adverse-effect level |
| LPRSA | Lower Passaic River Study Area |
| MOA | mode of action |
| MRL | minimal risk level |
| NBSA | Newark Bay Study Area |
| NCP | National Contingency Plan |
| NJ | New Jersey |
| NJDEP | New Jersey Department of Environmental Protection |
| NOAEL | no-observed-adverse-effect level |
| NTP | National Toxicology Program |
| NY | New York |
| OU | operable unit |
| PAH | polyaromatic hydrocarbon |
| PAR | Pathways Analysis Report |
| PbB | blood lead |
| PCB | polychlorinated biphenyl |
| PCDD/Fs | polychlorinated dibenzo(p)dioxins and furans |
| POD | point of departure |
| POTW | publicly owned treatment works |
| PPRTV | Provisional Peer Reviewed Toxicity Value |
| PRG | preliminary remediation goal |
| QAPP | Quality Assurance Project Plan |
| RAGS | Risk Assessment Guidance for Superfund |
| ReP | relative effects potency |
| RfD | reference dose |

| | |
|---|---|
| RI/FS | remedial investigation and feasibility study |
| RME | reasonable maximum exposure |
| RPF | relative potency factor |
| RSL | Regional Screening Level |
| SL | screening level |
| SQT | Sediment Quality Triad |
| STSC | Superfund Health Risk Technical Support Center |
| SV-CWCM | Small Volume Chemical Water Column Monitoring |
| SVOC | semi-volatile organic compound |
| TEC | toxicity equivalence concentration |
| TEF | toxic equivalency factor |
| TEQ | toxicity equivalence |
| Tierra | Tierra Solutions, Inc. |
| TPH | total petroleum hydrocarbon |
| UCL | upper confidence limit |
| UF | uncertainty factor |
| USACE | U.S. Army Corps of Engineers |
| USEPA | U.S. Environmental Protection Agency |
| VOC | volatile organic compound |

## Executive Summary

The Baseline Human Health Risk Assessment for the Newark Bay Study Area, referred to herein as the Baseline Human Health Risk Assessment (BHHRA), has been prepared as part of the Newark Bay Study Area (NBSA) remedial investigation/feasibility study (RI/FS). The BHHRA and RI/FS are being conducted by Glenn Springs Holdings, Inc. (GSH) on behalf of Occidental Chemical Corporation (the successor to Diamond Shamrock Chemicals Company [formerly known as Diamond Alkali Company]) pursuant to the Administrative Order on Consent (AOC) under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA Index 02-2004-2010; USEPA 2004a). The BHHRA meets the requirements of the AOC and National Contingency Plan (NCP) (USEPA 1990). This report describes the approach, methods, and assumptions used by GSH to conduct the BHHRA, in accordance with U.S. Environmental Protection Agency (USEPA) risk assessment guidance.

This BHHRA quantitatively evaluates both cancer risks and noncancer health hazards from exposure to chemicals of potential concern (COPCs) in Newark Bay. The BHHRA evaluates both current and future risks to children, adolescents, and adults in the absence of remedial actions and institutional controls. The BHHRA uses current USEPA policy and guidance as well as additional site data and analyses.

USEPA uses risk assessment as a tool to evaluate the likelihood and degree of chemical exposure and the possible adverse health effects associated with such exposure. The basic steps of the Superfund human health risk assessment process are the following: 1) Data Collection and Analysis to determine the nature and extent of chemical contamination in environmental media, such as sediment, water, and fish; 2) Exposure Assessment, which is an identification of possible exposed populations and an estimation of human chemical intake through exposure routes such as ingestion, inhalation, or skin contact; 3) Toxicity Assessment, which is an evaluation of chemical toxicity including cancer and noncancer health effects from exposure to chemicals; and 4) Risk Characterization, which describes the likelihood and degree of chemical exposure at a site and the possible adverse health effects associated with such exposure.

The primary purpose of a BHHRA is two-fold: (1) provide risk managers with an understanding of potential current and future human health risks in the absence of remediation or exposure controls, including uncertainties (USEPA 1989, 1991d), and (2) provide the public with information regarding human health risks. The BHHRA for the NBSA uses available data and information from recent site-specific studies in a risk-based framework to characterize potential human health risks currently and in the future, consistent with USEPA guidance (1989, 1991d, 2005a). The BHHRA has been performed in a manner consistent with the Revised Pathways Analysis Report (Revised PAR) for the NBSA (Battelle 2018), and addresses comments and revisions on draft Risk Assessment Guidance for Superfund (RAGS) Part D tables provided by USEPA, USEPA review of responses to comments on the draft RAGS Part D tables, and agreed-upon resolutions to draft RAGS Part D tables (USEPA 2017a, 2017b, 2017c, 2018a, 2018b, 2018c).

## ES.1 Summary of Key Findings

Consumption of fish or crab represents the primary source of risk to human health in the NBSA. For anglers who routinely consume their catch, the potential cancer risks exceed the NCP risk range of $10^{-6}$ to $10^{-4}$ used by USEPA to determine whether a site poses an unacceptable risk, and the noncancer hazards are above the goal of a noncancer hazard index equal to 1 (USEPA 1991d). These results are summarized below.

Fish Consumption

| | **Summary of Key Findings Angler/Sportsman - Fish Consumption (a)** | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | **RME** | | | | **CTE** | | | |
| | **Child** | **Adolescent** | **Adult** | **Combined Adult/Child** | **Child** | **Adolescent** | **Adult** | **Combined Adult/Child** |
| Cumulative Cancer Risk | 3E-04 | 3E-04 | 6E-04 | 8E-04 | 9E-06 | 1E-05 | 2E-05 | 3E-05 |
| Primary Contributors (b) | PCB-126: 31% (38% for all DL-PCBs)<br>2,3,7,8-TCDD: 28% (33% for all PCDD/Fs)<br>Non-DL PCBs: 18%<br>Arsenic, inorganic: 4%<br>Dieldrin: 3% | | | | PCB-126: 35% (43% for all DL-PCBs)<br>2,3,7,8-TCDD: 29% (33% for all PCDD/Fs)<br>Non-DL PCBs: 10%<br>Arsenic, inorganic: 6%<br>Dieldrin: 3% | | | |
| Cumulative Noncancer HI | 4E+01 | 3E+01 | 3E+01 | NA | 4E+00 | 2E+00 | 2E+00 | NA |
| Primary Contributors (b) | Non-DL PCBs: 32%<br>PCB-126: 20% (26% for all DL-PCBs)<br>2,3,7,8-TCDD: 19% (22% for all PCDD/Fs)<br>Methyl mercury: 6%<br>4,4'-DDD: 5% | | | | Non-DL PCBs: 31%<br>PCB-126: 20% (25% for all DL-PCBs)<br>2,3,7,8-TCDD: 17% (19% for all PCDD/Fs)<br>Methyl mercury: 8%<br>4,4'-DDD: 4% | | | |
| Noncancer Health Effects with HI>1 | Reproductive (DL compounds)<br>Whole-Body (non-DL PCBs)<br>Liver (pesticides)<br>Neurological (Methyl mercury) | | | | Reproductive (DL compounds) | | | |

Notes:

Shading indicates that the cumulative potential carcinogenic risk exceeds 10-4, or one or more target organ-specific hazard indices exceed one.

(a) Cumulative cancer risks differ only minimally based on the method for estimating toxicity equivalency (TEQ) concentration (Kaplan-Meier [KM] TEQ calculator vs. manual calculations); therefore, the results presented are those based on the KM TEQ calculator. See Section ES.2.3, Toxicity Assessment for a discussion of the two methods.
(b) Primary contributors for cancer risks include exposures for a total of 26 years (6 years as a child and 20 years as an adult). The evaluation of the noncancer hazard index for all chemicals based on exposures to a young child (7 years and younger), which represents the highest HI for all age ranges evaluated (i.e., child 1-<7 years, adolescent 7-<19 years, and adult >18 years).

Crab Consumption

| | Summary of Key Findings<br>Angler/Sportsman - Crab Consumption (a) | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | RME | | | | CTE | | | |
| | Child | Adolescent | Adult | Combined Adult/Child | Child | Adolescent | Adult | Combined Adult/Child |
| Cumulative Cancer Risk | 3E-04 | 3E-04 | 6E-04 | 8E-04 | 2E-05 | 2E-05 | 4E-05 | 5E-05 |
| Primary Contributors (b) | 2,3,7,8-TCDD: 52% (60% for all PCDD/Fs)<br>PCB-126: 19% (23% for all DL-PCBs)<br>Non-DL PCBs: 8%<br>Arsenic, inorganic: 6% | | | | 2,3,7,8-TCDD: 54% (63% for all PCDD/Fs)<br>PCB-126: 20% (24% for all DL-PCBs)<br>Arsenic, inorganic: 6%<br>Non-DL PCBs: 4% | | | |
| Cumulative Noncancer HI | 5E+01 | 3E+01 | 3E+01 | NA | 7E+00 | 4E+00 | 4E+00 | NA |
| Primary Contributors (b) | 2,3,7,8-TCDD: 33% (38% for all PCDD/Fs)<br>Copper: 26%<br>Non-DL PCBs: 14%<br>PCB-126: 12% (15% for all DL-PCBs) | | | | 2,3,7,8-TCDD: 33% (38% for all PCDD/Fs)<br>Copper: 26%<br>Non-DL PCBs: 14%<br>PCB-126: 12% (15% for all DL-PCBs) | | | |
| Noncancer Health Effects with HI>1 | GI Tract (Copper)<br>Reproductive (DL compounds)<br>Whole-Body (non-DL PCBs) | | | | GI Tract (Copper)<br>Reproductive (DL compounds) | | | |

Notes:

Shading indicates that the cumulative potential carcinogenic risk exceeds 10-4, or one or more target organ-specific hazard indices exceed one.

(a) Cumulative cancer risks differ only minimally based on the method for estimating toxicity equivalency (TEQ) concentration (Kaplan-Meier [KM] TEQ calculator vs. manual calculations); therefore, the results presented are those based on the KM TEQ calculator. See Section ES.2.3, Toxicity Assessment for a discussion of the two methods.
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Recreational and Worker Sediment and Surface Water Contact

The potential cumulative cancer risks and noncancer hazards for recreational receptors who visit the NBSA, including swimmers, waders, and boaters, and have direct contact with accessible surface sediment and surface water are within or below the NCP risk range and below the noncancer protection goal of 1 (USEPA 1991d) for both the RME and CTE scenarios. The same is true for workers who have direct contact with accessible surface sediment.

### ES.2 Summary of BHHRA

The BHHRA was conducted in accordance with USEPA's four-step risk assessment paradigm (USEPA 1989):

- Data evaluation and hazard identification
- Exposure assessment

- Toxicity assessment
- Risk characterization.

Each of the four steps is summarized below.

### ES.2.1 Data Evaluation and Hazard Identification

The BHHRA was based solely on validated data from the RI/FS program, which were collected in accordance with Quality Assurance Project Plans (QAPPs) approved by USEPA Region 2. These include:

- 41 accessible surface sediment samples (including field duplicates) from 39 nearshore and mudflat locations
- 131 near-surface (shallow) surface water samples from six locations in Newark Bay
- 95 samples (including duplicates) from five fish species (American eel, bluefish, striped bass, summer flounder, and white perch)
- 37 samples each of crab muscle only and crab hepatopancreas only.

All data were validated according to approved QAPPs, with nearly all of the data determined to be valid and acceptable for use in the BHHRA, as qualified. A total of 82 chemicals were identified as COPCs in one or more of these media based on a screening process that considered carcinogen status, essential nutrient status, frequency of detection, and comparison of maximum concentrations (the higher of the maximum detected concentration or maximum detection limit) to risk-based screening levels, similar to the Revised PAR. These included polychlorinated dibenzo(p)dioxins and furans (PCDD/Fs), polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons, various pesticides and inorganics, a few total petroleum hydrocarbon (TPH) ranges (PHC as gasoline and TPH [C9-C40][1]), volatile organic compounds (VOCs), and semivolatile organic compounds (SVOCs). An additional 56 chemicals were evaluated qualitatively in the uncertainty evaluation. The COPC screening process was designed to ensure that chemicals not identified as COPCs are only minor contributors to overall site risks and noncancer hazards.

### ES.2.2 Exposure Assessment

Newark Bay (the Bay) is a 6.3-square-mile enclosed embayment on the western side of the New York/New Jersey (NY/NJ) Harbor Estuary and is central to one of the most urbanized and industrialized areas in the United States. The Bay is adjacent to four large cities (Newark, Elizabeth, Bayonne, and Jersey City) and is fringed on its western side by port facilities, industrial facilities, and Newark Liberty International Airport. On its

[1] Traditionally, TPH is not assessed as a COPC in Superfund risk assessments, rather the individual chemical constituents that comprise TPH are evaluated. TPH ranges (PHC as gasoline and TPH [C9-C40]), were assessed throughout the steps of the BHHRA. Detailed information regarding screening values and toxicity factors have been addressed for TPH in Appendix B-1 and Section 5.1, respectively.

northern side, the Hackensack and Passaic Rivers flow into the Bay, while on the southern side, the Bay is connected to New York Harbor (NY) and Raritan Bay (NJ) through two tidal straits: Kill van Kull and Arthur Kill, respectively. The NBSA has been defined as the Bay and portions of key tributaries, including the Hackensack River, Arthur Kill, and Kill van Kull.

Human use of the NBSA is primarily industrial and commercial. Recreational use is more limited due to access limitations from the shoreline types (i.e., bulkhead, bridges, sheet piling, and mudflats) and surrounding urban/industrial/commercial land use. Access for recreation is through available public access areas (e.g., parks along the shoreline) and pleasure boating (i.e., launches from marinas inside and outside of the NBSA). Some consumption of fish and crab from the Bay has been reported, despite consumption advisories for certain fish species and a ban on the harvest and consumption of blue crab from the NBSA (Pflugh et al. 1999). People catch and consume fish and crab in the Bay, including species identified in the advisories. This has been reported along the Bayonne waterfront on the eastern side of the Bay; on the pilings of the Central Railroad of New Jersey/Newark Bay Bridge (also known as Old Bay Bridge), which was demolished in the 1980s; and at other piers, exposed rocky shorelines, pilings, and docks (Anglerweb.com, accessed April 27, 2017).

Potential receptors and exposure pathways identified for quantitative evaluation in the human health conceptual site model (HHCSM) for the NBSA include the following:

- Anglers/sportsmen who may be exposed via fish or shellfish[2] ingestion, dermal contact with sediment and surface water, and incidental ingestion of sediment and surface water
- Swimmers, waders, and boaters who may be exposed via dermal contact with sediment and surface water, and incidental ingestion of sediment and surface water
- Workers who may be exposed via dermal contact with sediment and incidental ingestion of sediment.

Potential exposure via inhalation of vapors in outdoor air as a result of volatilization of volatile and semivolatile organic compounds in sediment and surface water was shown to pose negligible risks (i.e., cancer risks < $10^{-6}$ and a noncancer hazards < 1) to all receptors by a quantitative screening-level evaluation conducted in a manner consistent with the BHHRA for the Lower Passaic River Study Area (LPRSA) (AECOM 2017); therefore, this pathway was excluded from the final cumulative risk estimates in the BHHRA. Briefly, for sediment, site-specific inhalation screening levels were developed using USEPA's *Soil Screening Guidance: User's Guide* (USEPA 1996a) assuming a cancer risk of $10^{-6}$ and a noncancer hazard quotient of 1. Screening-level cancer risks and hazard quotients were estimated based on the inhalation screening levels and the exposure point concentrations (EPCs; defined below) for all volatile or semi-volatile COPCs. All estimated cancer risks were below $10^{-6}$ (maximum of $1.4\times10^{-7}$) and all noncancer hazard

[2] While multiple shellfish may be present in Newark Bay, ingestion of shellfish is based solely on data for blue crab.

quotients were below 1 (maximum of 0.16). Additional information can be found in Section 4.1 and the complete documentation can be found in Appendix C-1.

For surface water, volatilization of COPCs was evaluated using a tiered approach. In Tier 1, ambient air concentrations were estimated for each volatile or semi-volatile COPC based on its EPC and a very conservative model for estimating evaporation from surface water and dispersion into a simple box model. For those chemicals for which the Tier 1 air concentration exceeded USEPA's residential air regional screening levels (RSLs), assuming a cancer risk of $10^{-6}$ and a noncancer quotient of 1, a Tier 2 air concentration was estimated based on the same EPC, but using a more realistic model for estimating evaporation from surface water and USEPA' screening-level air dispersion model AERSCREEN. The Tier 1 concentrations for chloroform, naphthalene, dioxin-like PCBs, and trichloroethylene exceeded the USEPA residential air RSLs; however, the Tier 2 concentrations for these COPCs were below the residential RSLs by at least an order of magnitude. Additional information can be found in Section 4.1 and the complete documentation can be found in Appendix C-2.

Exposure by ingestion of waterfowl or species other than fish and crabs (e.g., turtles, frogs) is not included in the quantitative risk assessment calculations. The types of waterfowl observed in the NBSA consume grass, not fish, which results in lower tissue concentrations. The New Jersey Division of Fish and Wildlife, Bureau of Law Enforcement has not observed anyone hunting in the NBSA (USEPA 2017a). Ingestion of waterfowl and animals other than fish/crabs at the NBSA appears to be minimal, especially relative to fish and crab consumption. Residential receptors are not included as an exposed population in the quantitative risk assessment calculations. The Newark Bay shoreline does not appear to support residential land use, because, although there are residences near the Bay, access to the Bay from the residential properties is limited by physical barriers such as steep slopes and rocks. There are observations that transient persons inhabit areas near and along the NBSA and may be exposed to NBSA sediment and surface water via incidental ingestion and dermal contact. While it is possible for short-term exposures to these receptors, the assumed exposures by long-term recreators and anglers/sportsmen (sediment, surface water, fish/crab consumption) are anticipated to be higher than those of a transient individual. Therefore, potential exposure via ingestion of species other than fish and crabs (the main exposure pathway of concern), and potential exposure of residential or transient receptors are discussed qualitatively in the uncertainty evaluation (Section 7).

Two exposure scenarios are evaluated in the BHHRA, consistent with USEPA (1992a) guidance: a reasonable maximum exposure (RME) scenario and a central tendency exposure (CTE) scenario. The intent of the RME scenario is to estimate a conservative exposure case that is above the average case but still within the range of possible exposures (USEPA 1989, 1992a). The CTE scenario uses average exposure parameters to calculate the average exposure of an individual. While risk management decisions are based on the RME scenario (USEPA 1989), these two scenarios provide risk managers with an estimated range of risks for the exposed population. The exposure assumptions for both scenarios are intended to reflect exposures under both current and future site uses. The fish and crab ingestion rates established by USEPA Region 2 (2012a, 2012b) for the Lower Passaic River Study Area (LPRSA) are used in this BHHRA. Exposure to fish and crab tissue, as well as accessible surface sediment and surface water,

is evaluated on a Bay-wide basis. In addition, the exposure-point concentration (EPC) for both the RME and CTE scenarios is the lower of the 95 percent upper confidence limit (95% UCL) of the arithmetic mean or the maximum concentration, consistent with USEPA guidance.

The BHHRA evaluated a "mixed fish" diet to account for the presence of multiple fish species in Newark Bay that may be consumed by anglers, which is assumed to comprise equal amounts (20%) of the five species collected as part of the RI/FS (American eel, bluefish, striped bass, summer flounder, and white perch). A supplemental analysis of individual fish species diets was included in the uncertainty evaluation. Similarly, the BHHRA evaluated crab muscle and hepatopancreas tissues combined, to account for the possibility that the crab is cooked before the hepatopancreas is removed. A supplemental analysis of a crab-muscle-only diet was included in the uncertainty evaluation (Section 7); risks/hazards for hepatopancreas only are presented in Appendices F and G. As discussed in Section 7, concentrations of PCDD/Fs, DL-PCBs, and non-DL PCBs are approximately 20 to 40 times higher in the hepatopancreas than in the muscle tissue alone. As a result, the concentrations of these chemicals in muscle and hepatopancreas combined are 7 to 10 times higher than in muscle alone. Finally, no cooking loss is considered in the RME scenario for both fish and crab consumption, which assumes that fat, pan drippings, and cooking juices are consumed. For the CTE scenario, cooking loss was included for fish consumption (insufficient data are available for crab consumption).

### ES.2.3 Toxicity Assessment

The toxicity criteria used in the BHHRA were selected according to USEPA (2003a; 2019b) guidance, including cancer and noncancer criteria for oral and dermal exposures. USEPA (2004b) default dermal absorption factors were used to adjust oral toxicity criteria for evaluating dermal exposure. In addition, USEPA's age-dependent adjustment factors were used to evaluate early-life exposures for chemicals believed to act by a mutagenic mode of action (USEPA 2005c, 2011b). Blood lead models, for adults and children, were used to evaluate potential exposure to lead in fish, crabs, and sediment. Specifically, USEPA's Integrated Exposure Uptake Biokinetic (IEUBK) model (Version 1.1, Build 11) was used to quantify potential exposures to lead for children younger than 7 years of age (USEPA 1994a, 1994b). The model developed by Bowers et al. (1994) was used to quantify exposures to lead for adolescent and adult receptors. The component of the model for soil is the same as that used in the USEPA Adult Lead Methodology (ALM) spreadsheet (USEPA 2017d). The ALM could not be used on its own because it does not account for exposure to lead via pathways other than soil (e.g., food).

For PCDD/Fs and dioxin-like (DL) PCBs (collectively referred to as dioxin-like compounds [DLCs]), cancer risks and hazard indices were estimated for the individual congeners, as well as in terms of a total toxicity equivalence (TEQ) for PCDD/Fs and PCBs (TEQ DF and TEQ PCB, respectively). The toxicity criteria for these compounds are based on the cancer and noncancer criteria for 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) and congener-specific toxicity equivalency factors (TEFs). The TEQ DF and TEQ PCB were calculated by two methods: (1) using USEPA's Kaplan-Meier (KM) calculator (Version 9.1; issued July 2014), and (2) manually based on the TEQ concentration for each congener. The first method is denoted as "based on KM TEQs" and the second method is denoted as "excluding KM TEQ." Cumulative risk/hazard

estimates accounting for all COPCs are also presented as “based on KM TEQ” and “excluding KM TEQ.” The calculations were performed both ways in order to employ the KM TEQ calculator (as was done for the BHHRA for the Lower Passaic River Study Area (LPRSA) (AECOM 2017), allow for determination of risks/hazards for individual congeners via the manual calculations, and then compare the results of the two methods. The remaining non-DL PCB congeners were evaluated as a group (Total non-DL PCBs) using toxicity criteria for PCBs (high risk) and Aroclor 1254 for cancer and noncancer effects, respectively. Cumulative risk/hazard estimates are presented based on KM TEQs, as well as based on TEQs calculated manually. As discussed further below, there is essentially no difference in the risk/hazard estimates between the two methods; however, the latter method allows for identification of the specific congeners that contribute most to the overall risk/hazard.

### ES.2.4 Risk Characterization Results

The estimated cancer risks were compared to the NCP risk range of $10^{-6}$ to $10^{-4}$, and estimated noncancer hazard quotients (HQs) and hazard indices (HIs) were compared to a goal of protection of 1 (USEPA 1991d). In addition, noncancer HIs greater than 1 were further evaluated on a target-organ-specific basis (USEPA 1989). Tables ES-1 through ES-4 below present the RME and CTE cumulative cancer risks and total noncancer hazard indices for all receptors and exposure pathways quantitatively evaluated in the BHHRA; additional details for the receptor age group with the highest potential cancer risk and noncancer hazard are presented following the tables.

**Table ES-1**
**Summary of Receptor/Exposure Pathway Cancer Risks for NBSA Baseline Human Health Risk Assessment (a)**
**Reasonable Maximum Exposure (RME)**

| Receptor Population | Age Group | Accessible Surface Sediment | Surface Water | Mixed Fish Diet (b) | Crab Muscle & Hepatopancreas |
|---|---|---|---|---|---|
| Angler/Sportsman | Child | *Pathways Incomplete* | | 3E-04 | 3E-04 |
| | Adolescent | 2E-06 | 8E-08 | 3E-04 | 3E-04 |
| | Adult | 4E-06 | 5E-08 | 6E-04 | 6E-04 |
| | Adult/Child (c) | 4E-06 | 5E-08 | 8E-04 | 8E-04 |
| Swimmer | Child | 1E-06 | 2E-07 | *Pathways Incomplete* | |
| | Adolescent | 2E-06 | 5E-07 | | |
| | Adult | 1E-06 | 9E-08 | | |
| | Adult/Child (c) | 2E-06 | 3E-07 | | |
| Wader | Child | 1E-06 | 3E-08 | | |
| | Adolescent | 2E-06 | 6E-08 | | |
| | Adult | 1E-06 | 1E-08 | | |
| | Adult/Child (c) | 2E-06 | 4E-08 | | |
| Boater | Child | *Pathways Incomplete* | | | |
| | Adolescent | 2E-06 | 3E-07 | | |
| | Adult | 4E-07 | 3E-07 | | |
| | Adult/Child (c) | *Not Applicable* | | | |
| Worker | Adult | 3E-06 | *Not quantified (d)* | | |

Notes:

Shading indicates that the cumulative potential carcinogenic risk exceeds 10-4.

(a) Cumulative cancer risks differ only minimally based on the method for estimating toxicity equivalency (TEQ) concentration (Kaplan-Meier [KM] TEQ calculator vs. manual calculations); therefore, the results presented are those based on the KM TEQ calculator.

(b) Mixed fish diet assumed to consist of equal fractions (20%) of American eel, bluefish, striped bass, summer flounder, and white perch.

(c) Cancer risks for adult and child age groups summed to yield 26-year total exposure duration.

(d) Workers are not expected to have contact with surface water during outdoor activities.

**Table ES-2**
**Summary of Receptor/Exposure Pathway Cancer Risks for NBSA Baseline Human Health Risk Assessment (a)**
**Central Tendency Exposure (CTE)**

| Receptor Population | Age Group | Accessible Surface Sediment | Surface Water | Mixed Fish Diet (b) | Crab Muscle & Hepatopancreas |
|---|---|---|---|---|---|
| Angler/Sportsman | Child | *Pathways Incomplete* | | 9E-06 | 2E-05 |
| | Adolescent | 4E-07 | 8E-09 | 1E-05 | 2E-05 |
| | Adult | 6E-07 | 5E-09 | 2E-05 | 3E-05 |
| | Adult/Child (c) | 6E-07 | 5E-09 | 3E-05 | 5E-05 |
| Swimmer | Child | 2E-07 | 4E-08 | *Pathways Incomplete* | |
| | Adolescent | 3E-07 | 1E-07 | | |
| | Adult | 2E-07 | 2E-08 | | |
| | Adult/Child (c) | 3E-07 | 5E-08 | | |
| Wader | Child | 2E-07 | 1E-08 | | |
| | Adolescent | 3E-07 | 7E-09 | | |
| | Adult | 2E-07 | 1E-09 | | |
| | Adult/Child (c) | 3E-07 | 6E-09 | | |
| Boater | Child | *Pathways Incomplete* | | | |
| | Adolescent | 3E-07 | 7E-08 | | |
| | Adult | 6E-08 | 4E-08 | | |
| | Adult/Child (c) | *Not Applicable* | | | |
| Worker | Adult | 3E-07 | *Not quantified (d)* | | |

Notes:

Shading indicates that the cumulative potential carcinogenic risk exceeds 10-4.

(a) Cumulative cancer risks differ only minimally based on the method for estimating toxicity equivalency (TEQ) concentration (Kaplan-Meier [KM] TEQ calculator vs. manual calculations); therefore, the results presented are those based on the KM TEQ calculator.

(b) Mixed fish diet assumed to consist of equal fractions (20%) of American eel, bluefish, striped bass, summer flounder, and white perch.

(c) Cancer risks for adult and child age groups summed to yield 12-year total exposure duration.

(d) Workers are not expected to have contact with surface water during outdoor activities.

**Table ES-3**
**Summary of Receptor/Exposure Pathway Noncancer Hazards for NBSA Baseline Human Health Risk Assessment (a)**
**Reasonable Maximum Exposure (RME)**

| Receptor Population | Age Group | Accessible Surface Sediment | Surface Water | Mixed Fish Diet (b) | Crab Muscle & Hepatopancreas |
|---|---|---|---|---|---|
| Angler/Sportsman | Child | *Pathways Incomplete* | | 4E+01 | 5E+01 |
| | Adolescent | 1E-01 | 2E-03 | 3E+01 | 3E+01 |
| | Adult | 1E-01 | 1E-03 | 3E+01 | 3E+01 |
| Swimmer | Child | 1E-01 | 8E-03 | *Pathways Incomplete* | |
| | Adolescent | 1E-01 | 1E-02 | | |
| | Adult | 3E-02 | 3E-03 | | |
| Wader | Child | 2E-01 | 1E-03 | | |
| | Adolescent | 1E-01 | 2E-03 | | |
| | Adult | 3E-02 | 4E-04 | | |
| Boater | Child | *Pathways Incomplete* | | | |
| | Adolescent | 1E-01 | 9E-03 | | |
| | Adult | 2E-02 | 9E-03 | | |
| Worker | Adult | 9E-02 | *Not quantified (c)* | | |

Notes:

Total hazard index presented. Shading indicates that one or more target organ specific hazard indices exceed one.

(a) Cumulative noncancer hazards differ only minimally based on the method for estimating toxicity equivalency (TEQ) concentration (Kaplan-Meier [KM] TEQ calculator vs. manual calculations); therefore, the results presented are those based on the KM TEQ calculator.

(b) Mixed fish diet assumed to consist of equal fractions (20%) of American eel, bluefish, striped bass, summer flounder, and white perch.

(c) Workers are not expected to have contact with surface water during outdoor activities.

**Table ES-4**
**Summary of Receptor/Exposure Pathway Noncancer Hazards for NBSA Baseline Human Health Risk Assessment (a)**
**Central Tendency Exposure (CTE)**

| Receptor Population | Age Group | Accessible Surface Sediment | Surface Water | Mixed Fish Diet (b) | Crab Muscle & Hepatopancreas |
|---|---|---|---|---|---|
| Angler/Sportsman | Child | *Pathways Incomplete* | | 4E+00 | 7E+00 |
| | Adolescent | 4E-02 | 5E-04 | 2E+00 | 4E+00 |
| | Adult | 4E-02 | 4E-04 | 2E+00 | 4E+00 |
| Swimmer | Child | 5E-02 | 4E-03 | *Pathways Incomplete* | |
| | Adolescent | 3E-02 | 6E-03 | | |
| | Adult | 1E-02 | 1E-03 | | |
| Wader | Child | 5E-02 | 4E-04 | | |
| | Adolescent | 3E-02 | 4E-04 | | |
| | Adult | 1E-02 | 1E-04 | | |
| Boater | Child | *Pathways Incomplete* | | | |
| | Adolescent | 3E-02 | 5E-03 | | |
| | Adult | 4E-03 | 3E-03 | | |
| Worker | Adult | 3E-02 | *Not quantified (c)* | | |

Notes:

Total hazard index presented. Shading indicates that one or more target organ specific hazard indices exceed one.

(a) Cumulative noncancer hazards differ only minimally based on the method for estimating toxicity equivalency (TEQ) concentration (Kaplan-Meier [KM] TEQ calculator vs. manual calculations); therefore, the results presented are those based on the KM TEQ calculator.

(b) Mixed fish diet assumed to consist of equal fractions (20%) of American eel, bluefish, striped bass, summer flounder, and white perch.

(c) Workers are not expected to have contact with surface water during outdoor activities.

Fish Consumption

The cumulative potential cancer risk for the RME combined adult/child angler/sportsman who routinely consumes a mixed diet of self-caught fish over a period of 26 years is $8\times10^{-4}$, regardless of TEQ approach. The primary contributors to the RME cumulative potential cancer risks are 2,3,7,8-TCDD, which contributes approximately 28% ($2\times10^{-4}$) (33% or 34% for all PCDD/Fs, depending on TEQ approach [$3\times10^{-4}$]); PCB-126, which contributes approximately 31% ($2\times10^{-4}$) (36 or 38% for all DL-PCBs, depending on TEQ approach [$3\times10^{-4}$]); and non-DL PCBs, which contributes approximately 18 or 19%, depending on TEQ approach ($1\times10^{-4}$). Minor contributors to the cumulative cancer risk include pesticides (approximately 5% [$4\times10^{-5}$) and inorganic arsenic (approximately 4% [$3\times10^{-5}$). Potential cancer risks associated with direct contact with accessible surface sediment or surface water are within or below the NCP risk range for the RME scenario.

The cumulative potential noncancer HI for the RME child angler who routinely consumes fish from the NBSA is 40, regardless of TEQ approach. As with excess cancer risk, the primary contributors to the cumulative potential HI are 2,3,7,8-TCDD, which contributes approximately 19% (HI of 8) (22% or 23% for all PCDD/Fs, depending on TEQ approach [HI of 10]); PCB-126, which contributes approximately 20% (HI of 9) (24% to 26% for all DL-PCBs, depending on TEQ approach [HI of 10]); and non-DL PCBs, which contribute approximately 32%, regardless of TEQ approach (HI of 10). The highest target-organ-specific HI is 20 for reproductive effects (DLCs), regardless of TEQ approach. The next highest target-organ-specific HI is 10 for whole-body effects (non-DL PCBs), regardless of TEQ approach. Liver (pesticides) and neurological effects (methyl mercury) are the only other target-organ-specific HIs greater than 1 (5 and 2, respectively).

The cumulative potential cancer risks for the CTE scenario for mixed fish diet are within the NCP risk range. For noncancer HIs, the only CTE target organ-specific HI greater than 1 (USEPA 1991d) is for reproductive effects (DLCs), where the HI is 2, regardless of TEQ approach.

Crab Consumption

The cumulative potential cancer risk for the RME combined adult/child angler/sportsman who routinely consumes a diet of self-caught crab muscle and hepatopancreas over a period of 26 years is also $8\times10^{-4}$, regardless of TEQ approach. The primary contributors to the RME cumulative potential cancer risks are 2,3,7,8-TCDD, which contributes approximately 52% ($4\times10^{-4}$) (59% or 60% for all PCDD/Fs, depending on TEQ approach [$5\times10^{-4}$]); PCB-126, which contributes approximately 19% ($2\times10^{-4}$) (23 or 24% for all DL-PCBs, depending on TEQ approach [$2\times10^{-4}$); and non-DL PCBs, which contributes approximately 8%, regardless of TEQ approach ($7\times10^{-5}$). Minor contributors to the cumulative cancer risk include inorganic arsenic (approximately 6% [$5\times10^{-5}$]) and pesticides (approximately 3% [$2\times10^{-5}$]). Potential cancer risks associated with direct contact with accessible surface sediment or surface water are within or below the NCP risk range for the RME scenario.

The cumulative potential noncancer HI for the RME child angler who routinely consumes muscle and hepatopancreas from the NBSA is 50, regardless of TEQ approach. As with excess cancer risk, the primary contributors to the cumulative potential HI are 2,3,7,8-TCDD, which contributes approximately 33% (HI of

20) (38% for all PCDD/Fs, regardless of TEQ approach [HI of 20]); copper, which contributes approximately 25% or 26%, depending on TEQ approach (HI=10), PCB-126, which contributes approximately 12% (HI of 6) (15% for all DL-PCBs, regardless of TEQ approach [HI of 7]); and non-DL PCBs, which contribute approximately 14%, regardless of TEQ approach (HI of 7). The highest target-organ-specific HI is 20 for reproductive effects (DLCs), regardless of TEQ approach. The next highest target-organ-specific HIs are 10 for GI tract (copper) and 7 for whole-body effects (non-DL PCBs), regardless of TEQ approach. The remaining target-organ-specific HI are equal to or less than 1.

The cumulative potential cancer risks for the CTE scenario for a crab muscle and hepatopancreas diet are within the NCP risk range. For noncancer HIs, the only CTE target organ-specific HI greater than 1 (USEPA 1991d) is for reproductive effects (DLCs), where the HI is 4, regardless of TEQ approach.

Direct Contact with Sediment and Surface Water

Cumulative potential cancer risks and noncancer HIs associated with direct contact with accessible surface sediment and surface water in the NBSA while angling, swimming, wading, or boating, are within or below the NCP risk range of $10^{-6}$ to $10^{-4}$ and below the noncancer protection goal of a HI of 1 (USEPA 1991d).

Lead Risk Characterization

Lead was identified as a COPC in accessible surface sediment and blue crab muscle/hepatopancreas tissue. For all receptors, PbBs were compared to 5 µg/dL, which is USEPA's target blood lead level used in the ALM spreadsheet (USEPA 2017d) and subsequent ALM guidance (USEPA 2017h), and consistent with CDC's current reference value for lead (CDC 2012). In addition, preliminary remediation goals (PRGs) were calculated for sediment and crab tissue using the same adult and adolescent-specific exposure parameters and a target PbB of 5 µg/dL.

The predicted PbBs for children <7 years of age who may be exposed to lead via crab consumption are less than 5 µg/dL for >99.9% of the population. Similarly, for adolescents and adults who are exposed to lead via crab consumption and direct exposure to accessible surface sediment, the predicted PbBs are below 5 µg/dL and crab tissue and sediment concentrations are below the calculated PRGs. The predicted PbBs for children <7 years of age who may be exposed to lead via direct contact with accessible surface sediment while swimming or wading are less than 5 µg/dL for >99.8% of the population. The predicted PbBs for adolescents and adults who may be exposed to lead via direct contact with accessible surface sediment while swimming, wading, or boating are below 5 µg/dL and sediment concentrations are below the calculated PRGs. The predicted PbB for adult workers who may be exposed to lead via direct contact with accessible surface sediment is below 5 µg/dL and sediment concentration is below the calculated PRG.

Taken together, these results indicate that lead in crab tissue and accessible surface sediment does not represent a hazard to an angler/sportsman. Similarly, lead in accessible surface sediment does not represent a hazard to a recreational swimmer, wader, or boater; nor an adult worker.

ES.2.5 Identification of Potential Chemicals of Concern

Potential COCs were identified in cases where the potential cumulative cancer risk or noncancer HI for a receptor exceeds $10^{-4}$ or 1, respectively. In these cases, potential COCs were any COPC with an individual pathway cancer risk greater than $10^{-6}$ or noncancer HI greater than 0.1. The following table summarizes the potential COCs for the RME scenario (no potential COCs were identified for surface water for either the RME or CTE scenario).

| Potential COC | Accessible Surface Sediment | Mixed Fish Diet | Crab Muscle and Hepatopancreas |
|---|---|---|---|
| **Dioxin-like Compounds** | | | |
| 2,3,7,8-TCDD | | X | X |
| 1,2,3,7,8-PeCDD | | X | X |
| 1,2,3,6,7,8-HxCDD | | X | |
| 2,3,7,8-TCDF | | X | X |
| 1,2,3,7,8-PeCDF | | X | X |
| 2,3,4,7,8-PeCDF | | X | X |
| 1,2,3,4,7,8-HxCDF | | X | X |
| 1,2,3,6,7,8-HxCDF | | X | X |
| Total PCDD/Fs (excluding KM TEQ) | | X | X |
| Total PCDD/Fs (based on KM TEQ) | | X | X |
| PCB-77 | | X | X |
| PCB-105 | | X | X |
| PCB-118 | | X | X |
| PCB-126 | | X | X |
| PCB-156/157 | | X | X |
| PCB-167 | | X | |
| PCB-169 | | X | X |
| Total DL-PCBs (excluding KM TEQ) | | X | X |
| Total DL-PCBs (based on KM TEQ) | | X | X |
| **Non-DL PCBs** | | | |
| Total Non-DL PCBs | | X | X |
| **PAHs** | | | |
| Benzo(a)pyrene | | X | |
| Dibenz(a,h)anthracene | | X | |
| **Pesticides & Organics** | | | |
| 2,4'-DDD | | X | |
| 4,4'-DDD | | X | X |
| 4,4'-DDE | | X | X |
| Chlordane, alpha (cis) | | X | |
| Dieldrin | | X | X |
| Heptachlor epoxide, cis- | | X | X |
| Heptachlor epoxide, trans- | | | X |
| Nonachlor, trans- | | X | X |
| Pyridine | | X | X |
| **Inorganics** | | | |
| Arsenic, inorganic | X | X | X |

| Potential COC | Accessible Surface Sediment | Mixed Fish Diet | Crab Muscle and Hepatopancreas |
|---|---|---|---|
| Cadmium | | | X |
| Cobalt | | X | X |
| Copper | | X | X |
| Mercury | | X | X |
| Methyl Mercury | | X | X |

### ES.3 Conclusions

**Fish and Crab**

Consumption of self-caught fish or crab from the NBSA presents the primary source of potential risk to human health. For the RME scenario, which is intended to represent a conservative exposure case that is above the average case but still within the range of possible exposures, the potential cancer risk and noncancer hazards to anglers/sportsman who are assumed to routinely consume their catch (34.6 g/day for an adult and 11.5 g/day for a child for fish, or 21 g/day for an adult and 7 g/day for a child for crab, over a period of 26 years) exceed the NCP risk range of $10^{-6}$ to $10^{-4}$ and a noncancer protection goal of an HI of 1 (USEPA 1991d). The RME cancer risk for the combined adult/child angler/sportsman is $8\times10^{-4}$ for both fish and crab consumption, and the noncancer HIs for the child angler/sportsman are 40 for fish consumption and 50 for crab consumption.

For the CTE scenario, which is based on average exposure levels (3.9 g/day for an adult and 1.3 g/day for a child for fish, or 3 g/day for an adult and 1 g/day for a child for crab, over a period of 12 years), the potential cancer risks for the combined adult/child angler/sportsman who consumes fish or crab from the NBSA are within the NCP risk range; however, noncancer HIs for the child angler/sportsman are above the noncancer protection goal of 1 (USEPA 1991d) (i.e., 4 for fish consumption and 7 for crab consumption).

The primary COPCs for fish and crab ingestion are 2,3,7,8-TCDD, PCB-126, and non-DL PCBs, with some pesticides, inorganic arsenic, and/or methyl mercury also contributing to the cumulative risks/hazards for both the RME and CTE scenarios.

As discussed in Section 7.3.3, there is considerable uncertainty in the TEFs for DL compounds, particularly for some of the DL-PCBs. Consistent with USEPA (2010a), a sensitivity analysis was conducted to illustrate the impact of the TEFs on the overall risk estimates and percent contribution of individual congeners or groups of congeners. For all congeners except 2,3,7,8-TCDD, the lower- and upper-bound TEFs were the $10^{th}$ and $90^{th}$ percentiles from *in vitro* and *in vivo* studies included in the relative effects potency (ReP) database (USEPA 2010a). The TEF for 2,3,7,8-TCDD remains constant in all scenarios. Accordingly, while the estimated risk for 2,3,7,8-TCDD remains constant, the contribution to risk can change, as well as the relative contribution of all PCDD/Fs, all DL-PCBs, and all PCBs (non-DL and DL-PCBs). For example, for the combined adult/child angler/sportsman who consumes a mixed fish diet, the percent contribution for 2,3,7.8-TCDD increases from 28% to 44% when using the lower-bound TEFs, but decreases to only 1%

when using the upper-bound TEFs. Conversely, the percent contribution to overall risk for Total PCBs (DL-PCBs and Non-DL PCBs) increases from 37% when using lower-bound TEFs to 98% when upper-bound TEFs are used. Similarly, for crab muscle and hepatopancreas consumption, the percent contribution of 2,3,7,8-TCDD increases from 52% to 70% when using the lower-bound TEFs, but decreases to approximately 2% when using the upper-bound TEFs. The percent contribution to overall risk for Total PCBs (DL-PCBs and Non-DL PCBs) increases from 16% when using lower-bound TEFs to 96% when upper-bound TEFs are used (see Section 7.3.3).

The specific species or tissue type(s) that make up a fish or crab diet can influence the estimated risk, because some species or tissue types have been shown to have higher tissue burdens of bioaccumulative chemicals than others. Fillet data were collected for the following five fish species from the NBSA: American eel, bluefish, striped bass, summer flounder, and white perch. The estimated cancer risks associated with consumption of any combination of these fish species exceed the NCP risk range for the RME scenario, but not the CTE scenario. The estimated noncancer HIs exceed the noncancer protection goal of an HI of 1 (USEPA 1991d) for both the RME and CTE scenarios. The estimated cancer risks associated with consumption of crab muscle only are approximately a factor of 5 to 6 lower than for muscle and hepatopancreas combined, but remain above the NCP risk range for the RME scenario. For noncancer effects, the noncancer HIs for a muscle-only diet are also approximately a factor of 3 lower than for muscle and hepatopancreas combined, but remain above the noncancer goal of 1 (USEPA 1991d) even for the CTE scenario. The risks/hazards to consumers of the crab hepatopancreas only would be much higher than the risks for consumers of muscle only or combined muscle and hepatopancreas (see Section 7).

**Sediment and Surface Water**

The cumulative potential cancer risks and noncancer HIs associated with direct contact with accessible surface sediment and surface water in the NBSA while angling, swimming, wading, or boating are much lower than those associated with fish or crab consumption and are within or below the NCP risk range of $10^{-6}$ to $10^{-4}$ and below the noncancer protection goal of an HI of 1 (USEPA 1991d).

**Lead**

No adverse health effects are expected to be associated with exposure to lead in crab tissue, or accessible surface sediment for any NBSA receptors.

# 1. Introduction

The Baseline Human Health Risk Assessment for the Newark Bay Study Area, referred to herein as the Baseline Human Health Risk Assessment (BHHRA), has been prepared as part of the Newark Bay Study Area (NBSA) remedial investigation/feasibility study (RI/FS). The BHHRA and RI/FS are being conducted by Glenn Springs Holdings, Inc. (GSH), on behalf of Occidental Chemical Corporation (the successor to Diamond Shamrock Chemicals Company [formerly known as Diamond Alkali Company]) pursuant to the Administrative Order on Consent (AOC) under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA Index 02-2004-2010; USEPA 2004a). The BHHRA meets the requirements of the AOC and National Contingency Plan (NCP) (USEPA 1990). This report describes the approach, methods, and assumptions used by GSH to conduct the BHHRA, in accordance with U.S. Environmental Protection Agency (USEPA) risk assessment guidance (USEPA 1986a, 1989, 1991a, 1991c, 1991d, 2001a, 2003a, 2004b, 2005b, 2005c, 2009a, 2011, 2014). The BHHRA is also consistent with the Revised Pathways Analysis Report (Revised PAR) for the NBSA (Battelle 2018). This report addresses comments and revisions provided by USEPA, USEPA review of responses to comments, and agreed-upon resolutions (USEPA 2017a, 2017b, 2017c, 2018a, 2018b, 2018c, 2019e; USEPA and NJDEP. 2019).

USEPA uses risk assessment as a tool to evaluate the likelihood and degree of chemical exposure and the possible adverse health effects associated with such exposure. The basic steps of the Superfund human health risk assessment process are the following: 1) Data Collection and Analysis to determine the nature and extent of chemical contamination in environmental media, such as sediment, water, and fish; 2) Exposure Assessment, which is an identification of possible exposed populations and an estimation of human chemical intake through exposure routes such as ingestion, inhalation, or skin contact; 3) Toxicity Assessment, which is an evaluation of chemical toxicity including cancer and noncancer health effects from exposure to chemicals; and 4) Risk Characterization, which describes the likelihood and degree of chemical exposure at a site and the possible adverse health effects associated with such exposure.

## 1.1 Background on NBSA Baseline Risk Assessment Planning

Several documents have been prepared that support the BHHRA for the NBSA. These include:

- Risk Assessment Scoping, Newark Bay Study Area Remedial Investigation, Baseline Human Health/Ecological Risk Assessment Workshop (Arcadis 2011)
- Newark Bay Study Area Problem Formulation for Baseline Human Health and Ecological Risk Assessment (Tierra Solutions, Inc. [Tierra] 2013)
- Quality Assurance Project Plans (QAPPs) developed for field sampling programs, including sediment, surface water, and tissue chemistry (Tierra 2014a, 2014b, 2015b; AECOM 2012a)
- Newark Bay Study Area Reconnaissance Survey Report. Baseline Human Health and Ecological Risk Assessment (Tierra 2015a)
- Proposed Risk Assessment Field Sampling and Analysis Program – Newark Bay Study Area (Arcadis 2015)
- Final Newark Bay Study Revised Pathways Analysis Report (Battelle 2018)
- Conceptual Site Model, Newark Bay Study Area, Revision 3 (GSH 2019a)

In addition, the BHHRA has been conducted in accordance with USEPA risk assessment guidance, including but not necessarily limited to:

- Risk Assessment Guidance for Superfund (RAGS) – Human Health Evaluation Manual (Parts A through F) (USEPA 1989, 1991a, 1991c, 2001a, 2004b, 2009a)
- Human Health Evaluation Manual, Supplemental Guidance: "Standard default exposure factors (USEPA 1991b)
- Guidelines for Exposure Assessment (USEPA 1992a)
- Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites (USEPA 2002a)
- Human Health Toxicity Factors in Superfund Risk Assessments (USEPA 2003a)
- Guidelines for Carcinogen Risk Assessment and Supplemental Guidance for Assessing Susceptibility from Early-Life Exposures to Carcinogens (USEPA 2005b, 2005c)
- Exposure Factors Handbook (USEPA 2011a)[3]
- Human Health Evaluation Manual, Supplemental Guidance: Update of Standard Default Exposure Factors (USEPA 2014)
- ProUCL Version 5.1 Technical Guide. Statistical Software for Environmental Applications for Datasets with and without Nondetect Observations (USEPA 2015a)
- Regional Screening Levels (USEPA 2019a)

### 1.2 Organization of BHHRA

The BHHRA was conducted in accordance with USEPA's four-step risk assessment paradigm, as outlined above (USEPA 1989):

- Data evaluation and hazard identification
- Exposure assessment
- Toxicity assessment
- Risk characterization.

The BHHRA report is organized as follows to address each of these steps:

- Section 2 — Site Characterization
- Section 3 — Data Evaluation and Hazard Identification
- Section 4 — Exposure Assessment
- Section 5 — Toxicity Assessment
- Section 6 — Risk Characterization
- Section 7 — Uncertainty Evaluation
- Section 8 — Summary and Conclusions

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[3] The last complete edition of the Exposure Factors Handbook was published in 2011; however, since 2017, USEPA has published updates to select chapters. For simplicity, this document is referenced as USEPA (2011a).

- Section 9 — References.

Tables and figures for each section are presented at the end of the text. The USEPA's RAGS Part D tables are split between report tables and appendices as outlined below.

| RAGS Part D Table | BHHRA Table Number or Location | Table Title |
|---|---|---|
| Table 1 | Table 4-1 | Selection of Exposure Pathways |
| Table 2 | Tables 3-8 to 3-10 | Occurrence, Distribution, and Selection of Chemicals of Potential Concern |
| Table 3 | Tables 4-14 to 4-18 | Exposure Point Concentration Summary |
| Table 4 | Tables 4-2 to 4-11 | Values Used for Daily Intake Calculations |
| Table 5 | Table 5-1 | Non-Cancer Toxicity Data – Oral/Dermal |
| Table 6 | Table 5-2 | Cancer Toxicity Data – Oral/Dermal |
| Table 7 | Appendix F | Calculation of Chemical Risks and Non-Cancer Hazards |
| Table 9[a] | Appendix G | Summary of Receptor Risks and Hazards for COPCs |
| Table 10 | Appendix I | Risk Summary |

[a] RAGS Part D Table 8, Calculation of Radiation Cancer Risks, is not applicable to the NBSA.

# 2. Site Characterization

The Diamond Alkali Superfund Site, which borders the Passaic River (Figure 2-1), was added to the Superfund National Priorities List on September 21, 1984, because of contaminants present at the site and in the river. Four different operable units (OUs) are associated with the site today and are shown on Figure 2-1: the former manufacturing plant and surrounding properties at 80 and 120 Lister Avenue (OU1), the lower 8.3 miles of the Passaic River (OU2), the Newark Bay Study Area (NBSA; OU3), and the lower 17 miles of the Passaic River (OU4; USEPA 2016a). The NBSA is the focus of this report. As noted, GSH is conducting an RI/FS for the NBSA. The data and information necessary to complete the BHHRA have been collected.

## 2.1 Site Setting

Newark Bay (the Bay) is a 6.3-square-mile enclosed embayment on the western side of the New York/New Jersey (NY/NJ) Harbor Estuary. The Bay is adjacent to four large cities (Newark, Elizabeth, Bayonne, and Jersey City), and is fringed on its western side by port facilities, industrial facilities, and Newark Liberty International Airport. On its northern side, the Hackensack and Passaic Rivers flow into the Bay, while on the southern side, the Bay is connected to New York Harbor (NY) and Raritan Bay (NJ) through two tidal straits: Kill van Kull and Arthur Kill, respectively. The NBSA has been defined as the Bay and portions of key tributaries, including the Hackensack River, Arthur Kill, and Kill van Kull (Figures 2-1 and 2-2). The Passaic River is not included in the definition of the NBSA, because it is currently being investigated as a separate OU. However, investigations of the Passaic River and NBSA OUs are being conducted in a comparable manner and with careful consideration of their linkages for the purposes of CERCLA management decision making, and broader environmental management considerations (GSH 2019a).

Newark Bay is central to one of the most urbanized and industrialized areas in the United States. It has experienced more than two centuries of environmental degradation that is attributable to many factors, including shoreline and land development (U.S. Army Corps of Engineers [USACE], 2006), wetlands/habitat loss, garbage and sewage disposal, dredging and dredged material disposal, and releases of contaminants from a variety of sources and locations (Iannuzzi et al. 2002).

### 2.1.1 Site Background

The environmental history of the Bay parallels the development of the New York City metropolitan area. Most shipping and economic development in the 19th century clustered around Manhattan and Brooklyn, but as the pace of development quickened in the first half of the 20th century, the Bay eventually supplanted Manhattan as the primary port by mid-century. Over that period, approximately 80% to 90% of the pre-existing shoreline of the Bay was developed, and ecological habitats correspondingly diminished (Iannuzzi et al. 2002; USACE 2009). A mid-19th century bathymetric map (Hassler 1844) depicts a shallow Bay (controlling depth less than 10 feet) that was bordered on the west and north by extensive wetlands (GSH 2019a).

The Bay has been the site of myriad industries for more than two centuries (Meyers 1945; Cunningham 1954; Brydon 1974; Iannuzzi et al. 2002). The development of the port system required extensive land development, achieved through "reclamation" of the meadowlands (wetlands) along the Bay and the Hackensack River during the 20th century. As the area's population and industrial development grew, transportation needs increased, and a large network of roads, bridges, airports, and port facilities was constructed. Additional information regarding the history of the site can be found in the Remedial Investigation Report (GSH 2019b).

The NBSA is known to be contaminated with a wide variety of organic compounds and inorganic chemicals (i.e., polychlorinated biphenyls [PCBs], polychlorinated dibenzo-p-dioxins and furans [PCDD/PCDFs], polycyclic aromatic hydrocarbons [PAHs], pesticides, herbicides, semivolatile organic compounds [SVOCs], volatile organic compounds [VOCs], inorganics/metals, and other organic compounds). As conceptualized in Figure 2-1, there are many known sources of contaminants to the Bay, including:

- Industrial discharges
- Publicly owned treatment works (POTWs), combined sewer overflows (CSOs), storm sewers, and other non-point sources
- Spills, leaks, and accidental discharges from marine and industrial sources
- Atmospheric deposition and groundwater discharges
- Tributary inputs from each of the sources listed, and transport of re-mobilized legacy sediments from tributaries.

Existing contamination in the NBSA is primarily from historical and current sources from each of these categories, which in combination have been released over more than a century, paralleling the urban and industrial history of the Bay. The relative influence or importance of these various sources is not easily quantifiable, and likely varies depending on the geographic area, COC group, temporal fate and transport processes, and the depth of the contaminated sediment layer under consideration. Additional information regarding sources of contaminants in the Bay is provided in the Report on Investigation of Sources of Pollutants and Contaminants (Tierra 2006).

### 2.2 Human Use of the Bay

Human use of the NBSA is primarily industrial and commercial. Recreational use is more limited due to access limitations from the shoreline types (i.e., bulkhead, bridges, sheet piling, and mudflats) and surrounding urban/industrial/commercial land use. Access for recreation is through available public access areas (e.g., parks along the shoreline) and pleasure boating (i.e., launches from marinas inside and outside of the NBSA). The likely current and future human users of the NBSA include recreational users (waders, swimmers, and boaters), anglers/sportsmen, workers, and residents and transients. These populations may be exposed to contaminants through direct contact with near-shore sediments and/or surface water during recreational activities, such as fishing, boating, working, or wading. They may also incidentally ingest contaminants from sediment and/or surface water during these activities. The most significant pathway by which people may be exposed to contaminants in the NBSA is expected to be from consuming fish and/or

crab. Human use of the NBSA shoreline is depicted on Figure 2-3 and is categorized as follows (Tierra 2015a):

- Disturbed Uplands — 18%
- Undisturbed Uplands — 26%
- Industrial/Commercial — 36%
- Recreational — 12%
- Residential — 11%.

Monitoring and research since the mid-1970s have resulted in the State of New Jersey taking several steps, including consumption advisories, closures, and bans on fish sales, to limit the exposure of the fish-eating public to toxic contaminants in the Bay. Consumption advisories still exist today in the northeast region of New Jersey for certain fish species. Specifically, there is an advisory warning the general public against any consumption of any gizzard shad from the NBSA, and limited consumption of bluefish, summer flounder, Atlantic needlefish, and rainbow smelt (one meal per month), striped bass, white perch, American eel, (four meals per year), and white catfish (one meal per year) (NJDEP and NJDOH 2019). High-risk populations (includes infants, children, pregnant women, nursing mothers and women of childbearing age) are advised against eating any of these fish species. Harvest and consumption of blue crab from the NBSA is banned (NJDEP and NJDOH 2019).

Some consumption of fish and crab from the Bay has been reported, despite the advisories and ban (Pflugh et al. 1999). People catch and consume fish and crab in the Bay, including species identified in the advisories. This has been reported along the Bayonne waterfront on the eastern side of the Bay; on the pilings of the Central Railroad of New Jersey/Newark Bay Bridge (also known as Old Bay Bridge), which was demolished in the 1980s; and at other piers, exposed rocky shorelines, pilings, and docks (Anglerweb.com, accessed April 27, 2017).

# 3. Data Evaluation and Hazard Identification

The purpose of the data evaluation and hazard identification process is two-fold: (1) evaluate the nature and extent of chemicals present in environmental media in the NBSA, and (2) identify chemicals of potential concern (COPCs) for further evaluation in the quantitative risk assessment. This step entails compiling and summarizing the data relevant to the BHHRA and identifying COPCs via a series of screening steps.

## 3.1 Data Evaluation

Several programs to collect samples of various environmental media have been conducted within the NBSA, including surface sediment sample collection, surface water sample collection, and collection of fish and crab tissues (biota). The data evaluated as part of the BHHRA were collected in accordance with USEPA-approved QAPPs (Crab-Clam QAPP, Tierra 2014a; Fish QAPP, Tierra 2014b; Sediment Quality Triad [SQT] QAPP, Tierra 2015b; SV-CWCM QAPP, AECOM 2012a), and data reports for each element of the program have been prepared and submitted (Crab-Clam Data Report, GSH 2017a; Fish Data Report, Tierra 2017; SQT Data Report, GSH 2017b; Surface Water Report, AECOM 2019). The data sets evaluated as part of the BHHRA are described below.

Validation of the data was performed according to procedures specified in the applicable QAPPs. Validation qualifiers were assigned to data based on criteria in the applicable data validation guidelines. All data that qualified as usable for their intended purposes, including risk assessment, were used in the COPC selection process, following USEPA (1989) guidance. Data rejected during data qualification (R-qualified) were excluded from evaluation in the BHHRA; however, data that were non-detect (U-qualified) and estimated (J-qualified) were included. Tables containing all analytical data used in the BHHRA are included in Appendix A, as well as a summary of the data validation and findings with regard to data usability in the BHHRA. Data analysis was performed using R (R Core Team 2018) and Microsoft Excel.

### 3.1.1 Surface Sediment Data Set

The BHHRA includes surface sediment sample data from the following sampling programs between 2014 and 2015:

- Crab and Clam Sampling and Analysis Program (Crab/Clam) (September–October 2014)
- Sediment Quality Triad and Porewater Sampling and Analysis Program (SQT) (September 2015).

To assess the impact of direct human contact with sediment (dermal and incidental sediment ingestion), sediment samples at human-accessible points along the shoreline are evaluated. Accessible surface sample locations are defined in Table 2 of the SQT QAPP (Tierra 2015b; see Attachment A-5 to Appendix A). The BHHRA includes sediment sample data from 16 accessible locations from the Crab/Clam program (including 1 field duplicate for a total of 17 samples), and 23 accessible locations from the SQT program (including 1 field duplicate for a total of 24 samples) (see Figure 3-1). Additional sediment samples were collected in the Phase III Sediment Investigation; however, because none of the locations were considered accessible by USEPA, no samples from this investigation were evaluated as part of the BHHRA.

In accordance with the SQT QAPP (Tierra 2015b), each sediment sample was analyzed for contaminants, including polychlorinated dibenzo(p)dioxins and furans (PCDD/Fs), polychlorinated biphenyls (PCBs) (209 individual congeners and Aroclors), metals (including mercury, methyl mercury, hexavalent chromium, and titanium), semivolatile organic compounds (SVOCs), polycyclic aromatic hydrocarbons (PAHs), volatile organic compounds (VOCs), pesticides, herbicides, butyltins/organotins, total petroleum hydrocarbons (TPH), ammonia, phosphorus, sulfide, and cyanide.

In the accessible surface sediment samples, several metals were measured using two analytical methods: cadmium, copper, lead, mercury, nickel, and zinc were measured using both USEPA Method 6010 and USEPA Method 6020. The BHHRA uses only the results from USEPA Method 6020. Table 3-1 identifies the number of samples analyzed for each contaminant by sampling program and by analytical method. Table 3-2 identifies the specific sediment surface samples included in the COPC selection process (see Section 3.3).

In 2017, comparison of sediment chemistry results between split samples analyzed by USEPA and Tierra Solutions, Inc., (Tierra) indicated that Tierra's results for PCBs and PAHs in samples from the SQT and Crab/Clam programs appeared to be biased low (LBG 2017). At USEPA's request, the sediment samples were reanalyzed for PCBs and PAHs after implementing corrective action on the relevant analytical methods. The results of the reanalysis were considered comparable to USEPA's results. For the BHHRA, the original results for PCBs and PAHs in sediment samples were discarded, and the reanalyzed results were used in their place.

### 3.1.2 Surface Water Data Set

The BHHRA includes data from the Small Volume (SV) Chemical Water Column Monitoring (CWCM) Sampling Program, performed as part of the RI/FS for the Lower Passaic River Study Area (LPRSA) (AECOM 2019). SV-CWCM sample and data collection were conducted during five rounds of routine sampling (August 2011, February 2012, March 2012, June 2012, December 2012), and two high-flow sampling events, when the flow through Dundee Dam was greater than 3,000 cubic feet per second (February/March 2013, June 2013). Samples were collected at 17 locations throughout the Lower Passaic River Study Area; however, the BHHRA includes samples from only six locations within Newark Bay proper (see Figure 3-2). (The SV-CWCM also included a low-flow/spring tide sampling event; however, none of the samples collected during this event were collected from locations within Newark Bay, and they were therefore excluded from the BHHRA.) Samples were collected from each location at two depths: 3 feet from the surface and 3 feet from the bottom. To reflect likely human interaction with surface water (i.e., wading, swimming, or boating), the BHHRA evaluated only samples that were taken at a depth of 3 feet or less (depth rounded to a single significant figure).

The following table summarizes the number of Newark Bay locations and surface water samples collected during each sampling event.

| Sampling Event | Date | Number of Newark Bay Locations | Number of Samples at Depth ≤ 3 feet |
|---|---|---|---|
| Round 1 | August 2011 | 4 | 16 |
| Round 2 | February 2012 | 5 | 19 |
| Round 3 | March 2012 | 5 | 19 |
| Round 4 | June 2012 | 6 | 19 |
| Round 5 | December 2012 | 5 | 20 |
| High Flow 1 | February/March 2013 | 5 | 19 |
| High Flow 2 | June 2013 | 5 | 19 |

As documented in the SV-CWCM Report (Table 2-2), not all samples were analyzed for all contaminants. The following analytes were monitored in every event: PCDD/Fs, PCB congeners and homologs, mercury, cadmium, copper, lead, sulfide, and chloride. The following analytes were monitored in only three routine sampling events and one high-flow sampling event: SVOCs, VOCs, metals, titanium, methyl mercury, hexavalent chromium, butyltins, pesticides, cyanide, PAHs, ammonia, and total phosphorus.

For metals and methyl mercury, both total and dissolved-fraction concentrations were measured. Only the total concentration was included in the BHHRA. Hexavalent chromium was measured as a dissolved-fraction concentration only; therefore, the dissolved-fraction concentration was included in the BHHRA.

For three chemicals/groups of chemicals, samples were analyzed by two analytical methods, as documented in the QAPP (AECOM 2012a). The data for these analytes were handled as follows:

- PAHs were measured using USEPA Method 8270C (along with other SVOCs) and a GC/MS-SIM method, KNOX-ID-0016. The GC/MS-SIM method yields improved detection limits compared to USEPA Method 8270C. When both measurements were available for the same PAH in the same sample, the BHHRA included only the results from Method KNOX-ID-0016. Otherwise, when only one measurement was available for a given PAH in a given sample, that measurement was used regardless of method.
- The metals arsenic, beryllium, cadmium, chromium, cobalt, copper, lead, nickel, silver, thallium, and zinc were measured using USEPA Method 6020 and USEPA Method 200.8. Each sample was analyzed using only one of the two methods. Results were used in the BHHRA regardless of method.
- Hexachlorobenzene was measured using USEPA Method 8270C (along with other SVOCs) and a modified version of USEPA Method 1699 (along with other organochlorine pesticides). The modified version of USEPA Method 1699 results in improved detection limits; therefore, in samples analyzed using both methods, the BHHRA used only the results from modified USEPA Method 1699. No samples were analyzed using only USEPA Method 8270C, and there was one sample analyzed using only modified USEPA Method 1699.

Table 3-3 identifies the number of samples analyzed for each contaminant by sampling program and by analytical method. Table 3-4 identifies the specific surface water samples included in the COPC selection process (see Section 3.3).

### 3.1.3 Fish and Crab Tissue Data Set

The BHHRA includes data from the crab and fish tissue collection programs conducted in the NBSA. Fish sampling activities were conducted within the three Newark Bay geographic zones (north, central, and south) during three fish sampling events: fall 2014, spring/summer 2015, and spring 2016 (see Figures 3-3 through 3-5). Blue crab sampling activities were conducted in September and October of 2014. Blue crab samples were collected from 12 Intertidal Areas; further blue crab samples were collected from each of the three Newark Bay geographic zones (eight locations in North, eight locations in Central, and nine locations in South Newark Bay) (see Figure 3-6).

The BHHRA includes data only on fish species from which fillet samples were collected: American eel, bluefish, striped bass, summer flounder, and white perch. Blue crab tissue samples included separate muscle and hepatopancreas samples. Although no combined muscle/hepatopancreas samples were collected directly, combined muscle/hepatopancreas results were calculated mathematically from the separate muscle and hepatopancreas results for each analyte. Specifically, muscle and hepatopancreas samples collected at the same location at the same time are from the same crab. Based on precise weight measurements of hepatopancreas, muscle and carcass of 34 individual crabs from station 129 (all crabs in composite measured) and station 131 (five of the crabs in the composite measured), it was estimated that 74% of the combined tissue mass for each crab was composed of muscle, and 26% was composed of hepatopancreas (see Attachment A-6 to Appendix A). Accordingly, the combined value was a weighted average of the separate muscle and hepatopancreas values, with weight 0.74 for muscle and 0.26 for hepatopancreas. Although blue crab carcass samples were collected, these were not analyzed as part of the BHHRA, because they are not considered relevant to human ingestion patterns.

The following table summarizes the number of tissue samples, including field duplicates, collected for each fish species and crab tissue type.

| Matrix | Species | Tissue | Number of Samples | Number of Field Duplicates |
|---|---|---|---|---|
| Fish | American Eel | Fillet | 18 | 0 |
| Fish | Bluefish | Fillet | 18 | 0 |
| Fish | Summer Flounder | Fillet | 18 | 0 |
| Fish | White Perch | Fillet | 22 | 4 |
| Fish | Striped Bass | Fillet | 21 | 3 |
| Crab | Blue Crab | Hepatopancreas | 37 | 0 |
| Crab | Blue Crab | Muscle | 37 | 0 |

In accordance with QAPPs (Tierra 2014a, 2014b), fish and crab tissue samples were analyzed for contaminants that included PCDD/Fs, PCBs (as congeners and Aroclors), metals (including methyl mercury, mercury, and titanium), SVOCs (including phthalates and alkylated PAHs), lipids, percent moisture, pesticides (excluding toxaphene), and butyltins. In fish fillet and crab muscle/hepatopancreas samples, each analyte was measured using only one method. Table 3-5 identifies the number of samples for each analyte by analytical method and by fish species or crab tissue type. Table 3-6 identifies the specific fish and crab tissue samples included in the COPC selection process (see Section 3.3).

For all species and tissues, arsenic was analyzed as total arsenic. Because only the inorganic form of arsenic is considered particularly toxic to humans, speciation of inorganic/organic arsenic in fish and crab tissue was estimated as follows: 10% of total arsenic was assumed to be inorganic arsenic, and the remaining 90% was assumed to be organic arsenic (see Section 5.5.4 and Appendix J).

## 3.2 Hazard Identification

The main purpose of the hazard identification step is to identify COPCs as a subset of all chemicals detected in each medium (surface water, sediment, and fish/crab tissue). A goal of the hazard identification is to evaluate the chemical carcinogenicity and potential noncancer health effects associated with the chemical-specific toxicity values. The hazard identification step enables the chemicals detected in each medium to be divided into two groups:

1. Chemicals that have negligible potential for adverse effects to humans and therefore do not need to be evaluated further.

2. Chemicals that have potential for adverse effects to humans and therefore require further evaluation—these are the COPCs.

### 3.2.1 Summary Statistics

For purposes of COPC selection, data for each medium were summarized, including frequency of detection, minimum and maximum detected concentrations, and range of detection limits. Details of the approach used to summarize the data by medium and chemical are provided below. As noted previously, results that were rejected during data validation (flagged “R”) were not included in the data summary, because these data are not usable for risk assessment (USEPA 1989). Only a small percentage of the data were rejected, as discussed in the uncertainty evaluation (see Section 7).

**Treatment of co-eluting PCB congeners:** Several PCB congeners were identified by the analytical laboratories as co-eluting congeners (see Table 3-7). Results for these co-eluting congeners represent the combined concentration for all congeners in the co-eluting set. They are identified in tables of results using the list of all co-eluting congeners separated by forward slashes, e.g., “PCB-156/157” or “PCB-86/87/97/109/119/125.” In the sediment and biota (fish and crab) samples there were 30 instances of co-elution, while in the surface water samples there were 34 instances of co-elution. Only one instance of dioxin-like PCBs was reported as co-eluted in both groups, i.e. “PCB-156/157.”

**Treatment of non-dioxin-like PCBs:** Non-dioxin-like PCBs (non-DL PCBs) were not evaluated individually as possible COPCs. Rather, a total value for non-DL PCBs was calculated by summing the reported concentrations of all non-DL PCBs for each sample. For sediment and biota samples, total values for non-DL PCBs were obtained by summing 128 unique and 29 co-eluting non-DL PCBs congeners. For surface water samples, total non-DL PCBs values were obtained by summing 118 unique and 33 co-eluting non-DL PCBs. Non-detects were replaced by the limit of detection. Total non-DL PCB value was assigned qualifier J (estimated) if any of the non-DL PCBs had qualifier J or no qualifier; if all of the non-DL PCBs had qualifier U (non-detect), then the total non-DL PCB value also was assigned qualifier U.

**Toxicity equivalence values:** For 17 PCDD/Fs, and separately for 12 DL PCBs, toxicity equivalence (TEQ) values were computed for each sample using USEPA's Kaplan-Meier TEQ (KM TEQ) calculator (Version 9.1; issued July 2014). After the congener concentrations were entered into the calculator, the KM TEQ calculator implemented a series of steps to compute the total TEQ values for each sample. First, the program calculated the toxicity equivalence concentration (TEC) for each congener by multiplying the congener's concentration by its toxic equivalency factor (TEF). In the case of non-detects, the detection limit was multiplied by the TEF. Second, the program used these TECs to obtain the intermediate "mean" TEC for all congeners. Finally, the program computed the total TEQ by multiplying the intermediate "mean" TEC by the number of congeners in the calculation. Of the 12 DL PCBs, the total concentration for PCB-156 and PCB-157 was reported by the lab as a single result, i.e., "PCB-156/157." This in and of itself is not a problem because both congeners have the same TEF. However, the program does not recognize "PCB-156/167," only "PCB-156" or PCB-157." Therefore, the total concentration for co-eluted "PCB-156/157" congeners was listed under "PCB-156," and no data were listed under "PCB-157." As implemented in the KM TEQ calculator, if more than 50% of the KM TEQ is contributed by samples with U or J qualifiers, then the resulting KM TEQ is assigned qualifier J (estimated); otherwise, the resulting KM TEQ is assigned no qualifier. Appendix D-2 provides more details on application of USEPA's KM TEQ calculator. For the manually calculated TEQs, each individual congener exposure-point concentration (EPC) was calculated using ProUCL in the same manner as EPCs for the other COPCs (see Sections 4.4 and 6.3).

**Treatment of duplicates:** There were two field duplicates in the sediment data (one from Crab/Clam and one from SQT investigations), and seven field duplicates in the fish fillet data. There were no field duplicates in the surface water data or the blue crab tissue data. For COPC identification, field duplicate sample results were treated as independent samples.

**Minimum concentration**: The minimum reported concentration for each chemical across all samples was determined, along with its qualifier (i.e., "U" for non-detect, "J" for estimated detect, or no qualifier for measured detect).

**Maximum concentration**: The maximum reported concentration for each chemical across all samples was determined, along with its qualifier (i.e., "U" for non-detect, "J" for estimated detect, or no qualifier for measured detect).

**Location(s) of maximum concentration:** The location identifier(s) for samples with the maximum concentration were reported. In sediment and biota (fish/crab) data sets, the maximum concentration

occurred at multiple locations; all of these locations were reported as a list. For the fish data set, location was identified as the geographic zone within Newark Bay (North, South, or Central). For crab data, a general and a specific location were identified. The general location was the geographic zone within Newark Bay (North, South, or Central). The specific location was given by the station identifier for the 12 Intertidal Area locations where a station identifier was available, or by the specific sample number within each geographic zone for the 25 samples that did not have specific coordinate information for sample location. For surface water and sediment data sets, the locations were identified using the station identifiers for each data set.

**Detection frequency**: For each chemical, the percentage of samples in which the measured value was above the detection limit was reported.

**Range of detection limits**: For each chemical, the range of reported detection limits across samples was reported. Detection limit may vary from sample to sample. Importantly, the minimum detection limit does not necessarily occur in the same sample as the minimum reported concentration, and the maximum detection limit does not necessarily occur in the same sample as the maximum reported concentration.

The data summaries for accessible surface sediment, surface water, fish tissue, and crab tissue are presented in Tables 3-8 through 3-11 (RAGS Part D Tables 2.1 to 2.4).

## 3.3 Method for COPC Selection

COPCs for the human health assessment were determined from sediment, surface water, and fish/crab tissue. COPCs were identified through a process that involved (1) identification of compounds classified by USEPA as a known human carcinogen, (2) evaluation of detection frequency, (3) identification of essential nutrients, and (4) comparison of the maximum concentration to risk-based screening values. A summary of the screening process is provided in Figure 3-7. Each of the key steps is outlined below.

### 3.3.1 Carcinogen Status

Chemicals detected in the historical data classified by USEPA as known human carcinogens were retained as COPCs, regardless of frequency of detection or detected concentration. Known human carcinogen designations are based on USEPA's weight of evidence classifications provided by USEPA (2005b, 2019b, 2019d). In addition, those chemicals that were not detected in any sample, but have been classified as a known human carcinogen, were included in the uncertainty evaluation.

### 3.3.2 Frequency of Detection

Chemicals detected in less than 5% of the samples were eliminated from further consideration as COPCs unless identified as a known human carcinogen (see Section 3.3.1). However, those chemicals that were either (1) detected in less than 5% of the samples or (2) not detected in any sample, but had maximum concentrations (detect or non-detect value) above the risk-based screening value (see Section 3.3.4), were included in the uncertainty evaluation.

### 3.3.3 Essential Nutrient Status

Inorganic constituents considered to be “essential nutrients,” which are not likely to be toxic at anticipated environmental levels, were excluded from consideration as COPCs. These included calcium, chloride, magnesium, phosphorus, potassium, and sodium.

### 3.3.4 Toxicity (Risk-Based) Screening

The maximum concentrations of all constituents that were detected in greater than 5% of the samples, except for known carcinogens and essential nutrients, were screened against a hierarchy of risk-based values for soil, tap water, and fish tissue. If no screening level was available, a surrogate chemical was identified, if possible, based on similarity in physical and chemical structure. Constituents with maximum concentrations exceeding the risk-based screening values were identified as COPCs, while constituents with concentrations below the risk-based screening values were excluded from further analysis. Those chemicals without a risk-based screening value, and for which no surrogate chemical could be identified, were included in the uncertainty evaluation. Importantly, background and ambient conditions were not considered during the screening process; therefore, the COPCs identified during the screen may include constituents that are not consistent with industrial sources or those that are typical of background conditions.

For sediment samples, the risk-based screening values are based on the USEPA Regional Screening Levels (RSLs) for residential soils as of May 2019 (USEPA 2019a). These risk-based values are derived to correspond to either a $1\times10^{-6}$ cancer risk or a noncarcinogenic hazard quotient (HQ) of 0.1 to account for potential cumulative effects. They were developed using default, conservative exposure assumptions for an integrated adult/child receptor (for cancer-based values) or a child receptor (noncancer-based values) assuming exposure through ingestion, dermal contact, and/or inhalation of vapors and fugitive dust from soil. Because no screening values are available for sediment, the soil screening values serve as conservative criteria, because it is likely that the potential receptors will spend less time offshore in the intertidal areas of Newark Bay as compared to onshore recreational/residential areas. The screening values used for chemicals in sediment are included in Table B-1.1 of Appendix B.

For surface water samples, risk-based screening values used for comparison to maximum concentrations are based on the USEPA RSLs for tap water as of May 2019 (USEPA 2019a). Similar to the residential soil RSLs described above, the tap water RSLs correspond to either a $1\times10^{-6}$ cancer risk or a noncarcinogenic HQ of 0.1 (to account for potential cumulative effects), assuming exposure through ingestion, dermal contact, and/or inhalation of contaminants in tap water. They were developed using default, conservative exposure assumptions for an integrated child/adult receptor (for cancer-based values) or a child receptor (for noncancer-based values). The tap water RSLs are conservative criteria for surface water, because potential receptors would be exposed via incidental ingestion and less frequent dermal contact, as compared to residential use of tap water. The screening values for chemicals in surface water are included in Table B-1.1 of Appendix B.

The USEPA does not publish RSLs for fish or crab tissue; however, risk-based screening values can be calculated using USEPA’s RSL calculator. These values were derived assuming fish or crab ingestion by an

adult, assuming an ingestion rate of 54 g/day (USEPA 1991b), for cancer-based screening levels and a child ingestion rate of 18 g/day for the noncancer-based screening levels. This latter value reflects a modification to the adult ingestion rate based on body-weight differences between adults and children. As with sediment and surface water, the screening values for noncarcinogenic effects were decreased by a factor of 10 for the purpose of this toxicity screen (HQ of 0.1). The screening values for chemicals in fish and/or crab are included in Table B-1.1 of Appendix B.

USEPA's current residential RSL (USEPA 2019a) for lead in soil is 400 mg/kg, based on the regulatory target of no more than 5% of young children in a population having a blood-lead level exceeding 10 µg/dL. USEPA now recommends targeting blood lead levels below 10 µg/dL; in fact, USEPA (2017h) recommends updates to lead methodologies and includes examples targeting a blood level of 5 µg/dL. Therefore, as recommended by USEPA and NJDEP (2019), 200 mg/kg is used as the screening level for lead in sediment. This value is derived based on a pharmacokinetic model designed to predict the probable blood lead concentrations for children between 6 months and 7 years of age who have been exposed to lead through various sources (air, water, soil, dust, diet, and in utero contributions from the mother). A tap water RSL of 15 µg/L is also available for lead; however, this value is not health-based, but is equal to USEPA's action level for lead in drinking water (USEPA 2019a). Finally, to screen for lead in fish or crab tissue, the Food and Drug Administration (FDA) action level for lead in crustacea of 1.5 mg/kg was used (FDA 2007).

### 3.4 COPC Selection

The results of the COPC selection process are presented in Tables 3-8 through 3-11 for accessible surface sediment, surface water, and fish/crab tissue, respectively (RAGS Part D Tables 2.1 to 2.4). A "Y" in the second-to-last column indicates that a chemical was retained as a COPC; an "N" indicates that a chemical was not retained as a COPC; and "UNC" indicates that the chemical will be discussed in the uncertainty evaluation. The basis for this determination is provided in the last column.

Several known human carcinogens, as defined by USEPA (2019b, 2019d, 2005b), were detected in one or more media, and were retained as COPCs in those media:

- Arsenic, inorganic
- Benzo(a)pyrene
- Hexavalent chromium [Cr(VI)]
- Trichloroethylene.

Three other known human carcinogens (i.e., benzene, benzidine, and vinyl chloride) were not detected in any sample and are discussed further in the uncertainty evaluation.

Several chemicals detected in sediment, surface water, and/or biota samples (which are not known human carcinogens) were not identified as COPCs based on low frequency of detection (detected in fewer than 5% of the samples) and the maximum concentration (detect or non-detect) below the screening value. In surface water, four chemicals detected in fewer than 5% of the samples had a maximum concentration (detect or non-detect) greater than the screening level (1,2,4-trichlorobenzene, 1,4-dichlorobenzene, 2,4-

dinitrotoluene, and cyanide); therefore, these chemicals are discussed in the uncertainty evaluation (Section 7). Similarly, one chemical in fish tissue (antimony) and five chemicals in crab tissue (1,2-diphenylhydrazine, 3,3'-dichlorobenzidine, antimony, benzaldehyde, and benzo(j,k)fluoranthene) were detected in fewer than 5% of the samples but had maximum concentrations (detect or non-detect) greater than their respective screening values. These chemicals are also discussed in the uncertainty evaluation.

### 3.4.1 Summary of COPCs

A number of chemicals/chemical groups were identified as COPCs in one or more media, as summarized below and in Table 3-13:

- Accessible surface sediment: 56 chemicals/chemical groups
- Surface water: 49 chemicals/chemical groups
- Fish tissue: 43 to 52 chemicals/chemical groups, depending on species (American eel, bluefish, striped bass, summer flounder, white perch)
- Crab tissue: 55 to 67 chemicals/chemical groups, depending on tissue type (muscle, hepatopancreas).

It is important to note that, for consistency, if a chemical was identified as a COPC in any fish or crab tissue, it was retained as a COPC for all tissue types. Therefore, the COPC lists used in the BHHRA are identical for all types of biota and include a total of 71 chemicals/chemical groups. In total, 82 chemicals/chemical groups were identified as COPCs.

Finally, a total of 13 chemicals were identified as COPCs in biota, but not in sediment or surface water, as shown in Table 3-12. These chemicals were generally detected at low concentrations (less than 1 mg/kg in sediment and less than 1 µg/L in surface water). For some chemicals (e.g., pesticides, methyl mercury), their presence in biota may reflect bioaccumulation.

# 4. Exposure Assessment

The purpose of the exposure assessment is to estimate the magnitude of current and reasonably anticipated future human exposure to COPCs associated with the NBSA. A detailed Conceptual Site Model (CSM) has been developed (GSH 2019a) that describes the current understanding of the NBSA and inter-relationships between sources, fate and transport, contaminated media, and receptors at the NBSA. This overall NBSA CSM is used below as the basis for a Human Health Conceptual Site Model (HHCSM) that identifies the sources, media, and exposure pathways by which humans are potentially exposed to COPCs. The exposure assessment calculates the frequency, duration, and magnitude of exposures associated with complete pathways in the HHCSM. Exposures are affected by concentrations of COPCs in exposure media, as well as characteristics of the exposure location, and activities and behaviors of potentially exposed individuals.

A combination of site-specific data representing the conditions and local population in the NBSA and USEPA default input values are used in the calculations to estimate exposures. The outcome of the exposure assessment is exposure-point concentrations and subsequent human receptor intakes of COPCs for all complete exposure pathways associated with current and reasonably anticipated future human exposures at the NBSA. Consistent with USEPA guidance (USEPA 1992a), two exposure scenarios are evaluated that represent reasonable maximum exposure (RME) and central tendency exposure (CTE), recognizing that risk management decisions are based on the RME scenario (USEPA 1989). The same COPCs are evaluated for both the RME and CTE scenarios.

This section is organized as follows:

- Section 4.1 discusses the HHCSM for the NBSA, including the potentially affected media, and the pathways by which people may be exposed to site media (potential exposure scenarios).
- Section 4.2 presents the methods used to quantify potential exposures for each potential exposure scenario.
- Section 4.3 identifies the exposure parameters and values used to quantify potential exposures.
- Section 4.4 describes the approaches used to estimate EPCs for each medium.

## 4.1 Human Health Conceptual Site Model

Figure 4-1 presents the HHCSM, which identifies the sources, media, and exposure pathways by which humans are potentially exposed to COPCs at the NBSA. A complete exposure pathway generally consists of four elements:

1. Source and mechanism of chemical release
2. Retention or transport medium
3. Point of potential contact with the contaminated medium (i.e., exposure point)
4. Exposure route (e.g., ingestion).

For risks to human receptors to be present, all of these elements must exist; otherwise, the pathway is deemed incomplete (USEPA 1989). Therefore, human exposure pathways at the NBSA are identified based on consideration of the sources, releases, types, and locations of chemicals at the site; the likely

environmental fate (including persistence, partitioning, transport, and intermedia transfer) of these chemicals; and the location and activities of the potentially exposed populations. The receptors and exposure scenarios associated with future use are not expected to differ significantly from those being evaluated under the current use.

Primary sources of contamination include industrial point sources, non-point-source runoff, POTW overflows, CSOs, tributaries, and atmospheric deposition. Secondary contamination sources include sediment and surface water.

As shown in Figure 4-1, the media relevant to evaluating potential human health exposures for the NBSA are:

- Sediment and surface water
- Fish tissue
- Shellfish tissue[4]
- Ambient air
- Waterfowl, turtles, and other species in the NBSA.

Human use activities in the NBSA are limited based on the shoreline type (e.g., bulkhead, bridges, sheet). Table 4-1 (RAGS Part D Table 1) presents the selection of potentially exposed human populations and exposure pathways and provides the rationale for inclusion of each pathway using either quantitative or qualitative methods.

The human receptors that have the greatest potential to be exposed to COPCs at the NBSA include a recreational user (e.g., boater, swimmer, wader) and an angler/sportsman. The complete set of potentially exposed populations in the NBSA includes the angler/sportsman, recreational user (boater, swimmer, wader), resident, transient, and port/dock worker. Potential exposure routes include ingestion of fish and shellfish, dermal contact with surface water and sediment, incidental ingestion of surface water and sediment, and inhalation of vapors (via ambient air).

The most significant pathway by which people may be exposed to chemicals in the NBSA is expected to be from consuming contaminated fish and/or shellfish. Following discharge, chemicals can partition by becoming attached to sediment, or they can remain suspended (or dissolved) in the water column. Chemicals enter the human food chain via bioaccumulation in tissues of fish and shellfish exposed directly to chemicals in the water column, in sediments, and/or in the tissues of prey.

In Appendix C-1, a screening assessment for the inhalation of volatile and semivolatile organic COPCs from exposed NBSA sediments was conducted to determine whether this route of exposure should be included in the BHHRA. Consistent with the BHHRA for the Lower Passaic River Study Area (LPRSA) (AECOM 2017),

[4] While multiple shellfish may be present in Newark Bay, ingestion of shellfish is based solely on data for blue crab.

inhalation screening levels for Newark Bay were developed using USEPA's *Soil Screening Guidance: User's Guide* (USEPA 1996a) assuming a cancer risk of $10^{-6}$ and a noncancer hazard quotient of 1 and USEPA default exposure assumptions (e.g., exposure frequency of 350 days/year and exposure duration of 26 years). These screening levels were then used to estimate screening-level cancer risks and noncancer hazard quotients based on the exposure point concentrations (EPCs; defined in Section 4.4) for all of the volatile and semi-volatile COPCs in sediment. The maximum screening-level estimated cancer risk was $1.4\times10^{-7}$ and the maximum estimated hazard quotient was 0.16. Based on these results, the sediment volatilization pathway was excluded from the final cumulative risk estimates in the BHHRA.

Similarly, in Appendix C-2, the potential for exposure to volatile or semivolatile organic COPCs in surface water via inhalation of vapors in ambient air was evaluated in a manner consistent with the baseline human health risk assessment for the LPRSA (AECOM 2017). Specifically, volatilization of COPCs from surface water was evaluated using a tiered approach. The first tier was based on a very conservative USEPA model for estimating evaporation from surface water (worst-case evaporation from a pool) and dispersion into a simple box model assuming a regional estimate of average wind speed of 4.5 miles per hour, a cross-wind dimension (width of the box) of 1500m (based on the approximate average width of Newark Bay), and a box height of approximately 6 feet. The resulting ambient air concentrations were compared to USEPA residential air RSLs, assuming a cancer risk of $10^{-6}$ and a noncancer hazard quotient of 1. The Tier 1 air concentration estimate for chloroform, naphthalene, non-DL PCBs, and trichloroethylene exceeded the residential air RSL; therefore, these chemicals were further evaluated. The second tier was based on a more realistic USEPA model for estimating evaporation from surface water (based on lagoons and surface impoundments) and USEPA's current screening-level air dispersion model AERSCREEN. As shown in the appendix, the estimated annual average air concentrations for all four remaining COPCs were below their respective residential air RSLs, by at least an order of magnitude. Accordingly, the surface water volatilization pathway was not included in the final cumulative risk estimates in the BHHRA.

Exposures by ingestion of waterfowl or species other than fish and shellfish are not included in the quantitative risk assessment calculations. The New Jersey Division of Fish and Wildlife, Bureau of Law Enforcement has not observed anyone hunting in the NBSA (USEPA 2017a). In addition, the type of waterfowl observed in the NBSA consume grass, not fish, which would result in lower tissue concentrations. For these reasons, ingestion of waterfowl and animals other than fish/crabs is likely to be minimal. This topic is discussed further in the uncertainty evaluation (Section 7).

Residential receptors are not included as an exposed population in the quantitative risk assessment calculations. As described in the Problem Formulation (Tierra 2013), the Newark Bay shoreline does not appear to support residential land use, because, although there are residences near the Bay, access to the Bay from the residential properties is limited by physical barriers such as steep slopes and rocks. Limited residential areas were observed along the eastern shore of the Bay; these areas have either manmade or natural barriers to impede human access to the Bay. Surface water from the Bay is not used as a domestic water supply. Residents may contact surface water during activities near their homes, but this contact is expected to be minor. Potential risks to residential receptors relative to other receptors are discussed in the uncertainty evaluation (Section 7).

Transient persons are not included as an exposed population in the quantitative risk assessment calculations. Although transients have been observed in temporary makeshift shelters near the Passaic River (Proctor et al. 2002), adequate information on the exposure patterns for this population is not available and is difficult to collect. Therefore, the exposures of the transient people are addressed qualitatively in the uncertainty section of the BHHRA.

Potentially exposed human receptor populations and pathways at the NBSA that are included in the quantitative analysis of the BHHRA are shown in Figure 4-1.

## 4.2 Quantification of Potential Exposures

In this section, equations used to quantify potential COPC chronic daily intakes, and the exposure assumptions and parameters of the equations, are presented and discussed. Exposure assumptions are based on current and future land use, which is described in Section 2.2. Exposure assumptions and parameters are consistent with site conditions and use standard USEPA risk assessment approaches.

The calculated COPC chronic daily intake is expressed in units of milligram COPC per kilogram body weight per day (mg/kg-day). For COPCs that are noncarcinogenic, the chronic daily intake is averaged over the exposure duration (ED). For COPCs that are carcinogenic, the chronic daily intake is averaged over the assumed receptor's lifetime (70 years).

### 4.2.1 Estimating Potential Exposure to COPCs in Sediment

Multiple receptors may be exposed to COPCs in sediment via incidental ingestion and dermal contact. As noted previously, the potential exposure to volatile COPCs in sediment via inhalation is not of concern (see Appendix C-1). The following equations were used to estimate potential exposure to COPCs in sediment (USEPA 1989; 2004b; 2019a).

Intake (lifetime and chronic) following incidental ingestion of sediment (mg/kg-day):

$$Intake = \frac{C_s \times IR_{sed} \times FI \times EF \times ED \times RBA \times CF}{BW \times AT}$$

Where:

Intake = intake (mg/kg-day)

$C_s$ = exposure-point concentration – sediment (mg/kg sediment)

$IR_{sed}$ = ingestion rate of sediment (mg sediment/day)

FI = fraction from source (unitless)

EF = exposure frequency (days/year)

ED = exposure duration (year)

RBA = relative bioavailability factor (chemical-specific) (unitless)

CF = conversion factor (kg sediment/$10^6$ mg sediment)

BW = body weight (BW)

AT = averaging time (days)

Intake (lifetime and chronic) following dermal contact with sediment (mg/kg-day):

$$Intake = \frac{C_s \times SA \times AF \times EF \times ED \times ABSd \times FI \times CF}{BW \times AT}$$

Where:

Intake = intake (mg/kg-day)

$C_s$ = exposure-point concentration – sediment (mg/kg sediment)

SA = skin surface area ($cm^2$/day)

AF = adherence factor ($mg/cm^2$)

EF = exposure frequency (days/year)

ED = exposure duration (year)

ABSd = dermal absorption factor (chemical-specific) (unitless)

FI = fraction from source (unitless)

CF = conversion factor (kg sediment/$10^6$ mg sediment)

BW = body weight (BW)

AT = averaging time (days)

### 4.2.2 Estimating Potential Exposure to COPCs in Surface Water

Multiple receptors may be exposed to COPCs in surface water via incidental ingestion and dermal contact. As noted previously, the potential exposure to volatile COPCs in surface water via inhalation is not of

concern (see Appendix C-2). The following equations were used to estimate potential exposure to COPCs in surface water (USEPA 1989; 2004b).

Intake (lifetime and chronic) following incidental ingestion of surface water (mg/kg-day):

$$Intake = \frac{C_{wat} \times IR_{wat} \times FI \times EF \times ED}{BW \times AT \times CF}$$

Where:

Intake = intake (mg/kg-day)

$C_{wat}$ = exposure point concentration – surface water (µg/L water)

$IR_{wat}$ = ingestion rate of surface water (L water/hour)

FI = fraction from source (unitless)

ET = exposure time (hours/day)

EF = exposure frequency (days/year)

ED = exposure duration (year)

RBA = relative bioavailability factor (chemical-specific) (unitless)

CF = conversion factor ($10^3$ µg chemical/mg chemical)

BW = body weight (kg)

AT = averaging time (days)

EPA (2004b) guidance for calculating dose from dermal exposure to surface water differentiates between organic and inorganic chemicals. Dermally absorbed dose (lifetime and chronic) following dermal exposure to surface water (mg/kg-day):

*Inorganics*

$$DAD = \frac{DA_{event} \times SA \times EV \times EF \times ED}{BW \times AT}$$

Where:

DAD = dermally exposed dose (mg/kg-day)

$DA_{event}$ = absorbed dose per event (mg/cm$^2$-event)

SA = skin surface area (cm$^2$)

EV = event frequency (event/day)

EF = exposure frequency (days/year)

ED = exposure duration (years)

BW = body weight (kg)

AT = averaging time (days)

The dose absorbed per unit area per event (DAevent) is calculated as follows for inorganics or highly ionized organics:

$$DA_{event} = C_{wat} \times K_p \times ET \times CF_1 \times CF_2$$

Where:

$DA_{event}$ = absorbed dose per event (mg/cm$^2$-event)

$C_{wat}$ = exposure point concentration – surface water (µg/L water)

$K_p$ = permeability constant (cm/hr) (chemical-specific)

ET = exposure time (hours/event)

$CF_1$ = conversion factor (L/1000 cm$^3$)

$CF_2$ = conversion factor (mg/1000 µg)

The $DA_{event}$ for organics is calculated as follows:

If ET ≤ t*

$$DA_{event} = 2\,FA \times Kp \times C_{wat} \times CF \sqrt{\frac{6\,tau_{event} \times ET}{\pi}}$$

If ET > t*

$$DA_{event} = FA \times K_p \times C_{wat} \times CF \left[\frac{ET}{1+B} + 2\,tau_{event}\left(\frac{1+3B+3B^2}{(1+B)^2}\right)\right]$$

Where:

$DA_{event}$ = absorbed dose per event (mg/cm$^2$-event)

FA = faction absorbed water

Kp = dermal permeability constant (cm/hour) (chemical-specific)

$C_{wat}$ = exposure-point concentration – surface water (µg/L water)

$tau_{event}$ = lag time per event (hour/event) (chemical-specific)

ET = exposure time (hours/event)

t* = time to steady state (hour); 2.4 ×$tau_{event}$

CF = conversion factor (L/1000 cm$^3$)

#### 4.2.3 Estimating Potential Exposure to COPCs in Fish/Shellfish Tissue

The angler/sportsman may be exposed to COPCs in fish or shellfish tissue via ingestion. The following equation was used to estimate potential exposure to COPCs in fish tissue (USEPA 1989).

Intake (lifetime and chronic) following fish/shellfish ingestion:

$$Intake = \frac{C_t \times IR \times (1 - Loss) \times FI \times EF \times ED \times CF}{BW \times AT}$$

Where:

Intake = intake (mg/kg-day)

$C_t$ = exposure-point concentration – tissue (mg/kg tissue)

$IR_{sed}$ = ingestion rate of fish/shellfish (g tissue/day)

FI = fraction from source (unitless)

EF = exposure frequency (days/year)

ED = exposure duration (year)

CF = conversion factor (kg tissue/$10^3$ g tissue)

BW = body weight (BW)

AT = averaging time (days)

## 4.3 Receptor- and Chemical-Specific Exposure Parameters

This section presents the receptor- and chemical-specific exposure parameters that are inputs to the equations presented in Section 4.2 to quantify potential intake of COPCs by each exposure pathway and human receptor population identified for the NBSA. Consistent with USEPA guidance (USEPA 1992a), two exposure scenarios are evaluated in the BHHRA that represent reasonable maximum exposure (RME) and central tendency exposure (CTE), even though risk management decisions are based on the RME scenario (USEPA 1989). In risk calculations, the difference between these two scenarios is reflected in different exposure parameter values. The intent of the RME is to estimate a conservative exposure case that is above the average case but still within the range of possible exposures (USEPA 1989, 1992a). The CTE uses average exposure parameters to calculate the average exposure of an individual.

The values used for each of the RME and CTE exposure parameters are presented in Tables 4-2 to 4-11 (RAGS Part D Tables 4.1 to 4.10); chemical-specific parameters are presented in Tables 4-12 and 4-13. The exposure parameter values are intended to represent both current and reasonably anticipated future conditions at the NBSA. The receptors and exposure scenarios associated with future use are not expected to differ significantly from those being evaluated under current use.

A description of each receptor evaluated quantitatively in the BHHRA is provided below, followed by discussions of receptor- and chemical-specific exposure parameters.

### 4.3.1 Angler/Sportsman Definition

The angler/sportsman is defined as an adult or adolescent catching and consuming a variety of fish (i.e., American eel, bluefish, striped bass, summer flounder, and white perch) or shellfish (i.e., blue crab) from the banks of the NBSA or a boat on the NBSA for recreational purposes. In spite of the “eat none” fish/crab consumption advisories (NJDEP and NJDOH 2019), the collection and consumption of fish and shellfish from the NBSA has been documented (Burger et al. 1999; Burger 2002; NJDEP 2002; Pflugh et al. 1999). Also, any fishing or crabbing that occurs along the shore could result in direct contact with both surface water and sediment. Therefore, for the angler/sportsman, the pathways quantitatively evaluated include fish and crab ingestion, dermal contact with sediment and surface water, and incidental ingestion of sediment and surface water. Inhalation may occur if activities occur in areas where volatiles are present in sediment or surface water; however, this pathway is not considered further in the BHHRA, because the inhalation pathway risks are negligible (i.e., cancer risks less than $10^{-6}$ and noncancer hazard quotients less than 1; see Appendices C-1 and C-2).

Anglers are assumed to share self-caught fish and/or crab with family members (i.e., children 1 to <7 years of age). Young children are expected to rarely accompany the family member who is fishing. Exposures

would be much less than those experienced by children who visit the Bay to wade or swim. Therefore, the exposure of a child to sediment and surface water is not evaluated under the angling scenario.

An evaluation of subsistence fishing is not included in the BHHRA, because there is no evidence of individuals who rely solely on their daily catch to subsist.

#### 4.3.2 Swimmer Definition

Recreational use associated with the NBSA includes boating, wading, and swimming, as well as walking or playing along the shore on exposed sediment. Thus, exposure to sediment and surface water is expected. Swimming does occur in Newark Bay. However, the exposure frequency and duration for swimming are reasonably assumed to be relatively low, both currently and in the future, due to the deterrents to swimming in the Bay. These include the presence of trash and debris, pathogenic contamination, and ship traffic. In addition, based on the NBSA Reconnaissance Survey Report (Tierra 2015a), only 12% of the shoreline was categorized as “recreational.” Of the over 200 individuals observed during the five-day survey, the main recreational activity observed was fishing, followed by jogging or walking along the shoreline (including playing with dogs). No one was observed swimming (Tierra 2015a). While this survey is limited—a five-day period during a single season—the results support the conclusion that swimming in the NBSA occurs relatively infrequently. Swimmers may experience incidental ingestion of surface water and may contact sediment while entering and leaving the Bay from the banks of the water. Inhalation may occur if activities are in areas where volatiles are present in sediment or surface water; however, this pathway is not considered further in the BHHRA, because the inhalation pathway risks are negligible (i.e., cancer risks less than $10^{-6}$ and noncancer hazard quotients less than 1; see Appendices C-1 and C-2).

#### 4.3.3 Wader Definition

Families visiting parks along the banks or wading down by the Bay to bird watch may contact surface water and sediment along the banks. Inhalation may occur if activities are in mudflat areas and volatiles are present in sediment or surface water; however, this pathway is not considered further in the BHHRA, because the inhalation pathway risks are negligible (i.e., cancer risks less than $10^{-6}$ and noncancer hazard quotients less than 1; see Appendices C-1 and C-2).

#### 4.3.4 Boater Definition

The potential exists for recreational boating, including kayaking, to occur in the Bay. It is assumed that the boater’s potential for exposure to Bay sediment is greatest while boating in small crafts such as sculls, kayaks, or canoes. Docks are typically used, and boaters are expected to remain in their boats, but boaters may occasionally contact sediment when wading is necessary. Young children (<7 years old) are not expected to participate in boating activities on the Bay; any such exposure would be rare and much less than that experienced by young children visiting the Bay specifically to wade or swim. Therefore, a young child boater scenario is not evaluated. Inhalation may occur if activities are in areas where volatiles are present in sediment or surface water; however, this pathway is not considered further in the BHHRA,

because the inhalation pathway risks are negligible (i.e., cancer risks less than $10^{-6}$ and noncancer hazard quotients less than 1; see Appendices C-1 and C-2).

### 4.3.5 Worker Definition

Workers may be assigned to collect shoreline trash or perform other work that leads to contact with sediment along the Bay. It is assumed that workers are adults (>18 years of age). Contact with surface water is not typically expected to occur. Inhalation may occur if activities are in mudflat areas and volatiles are present in sediment; however, this pathway is not considered further in the BHHRA, because the inhalation pathway risks are negligible (i.e., cancer risks less than $10^{-6}$ and noncancer hazard quotients less than 1; see Appendix C-1).

### 4.3.6 Fish and Crab Consumption Exposure Parameters

As explained in Section 4.1, the most significant pathway by which people may be exposed to chemicals in the NBSA is expected to be from consuming contaminated fish and/or shellfish (crab). The following subsections discuss exposure parameters used to calculate COPC intakes by the fish and crab consumption pathways. These parameters include fish and crab ingestion rates, fractions of fish and crab consumed that are from the NBSA, and the amount of chemical lost during the cooking process.

#### *4.3.6.1 Fish Ingestion Rate*

Fish and crab ingestion rates used in the BHHRA were developed as part of the LPRSA BHHRA (USEPA 2012a, 2012b). The ingestion rate assumes that the fish are caught only from the NBSA. It is assumed that ingestion of fish from local sources will be the main source of fish consumption for the angler/sportsman. For consumption of fish, the analysis of ingestion rates was based on data for anglers/sportsmen from the following sources:

- Exposure Factors Handbook (USEPA 2011a)
- Two surveys conducted for the Newark Bay Complex (Burger 2002, May and Burger 1996)
- A survey conducted for Barnegat Bay, an estuary on the New Jersey shore (Burger et al. 1998)
- The New Jersey Household Fish Consumption Survey (CPIP and NJMSC 1993)
- A statewide angler survey conducted in New York (Connelly et al. 1992).

Based on USEPA's evaluation of these studies, estimates of adult fish ingestion rates were derived for both the RME and CTE adult angler/sportsman using the $90^{th}$ and $50^{th}$ percentile ingestion rates, respectively, from the Burger (2002) and Connelly et al. (1992) data. The adult fish ingestion rates derived (USEPA 2012b) and used in the NBSA BHHRA are:

- RME adult angler/sportsman = 34.6 g/day; this rate is the average of the $90^{th}$ percentile value of the two studies
- CTE adult angler/sportsman = 3.9 g/day; this rate is the average of the $50^{th}$ percentile value of the two studies.

Fish ingestion rates for the adolescent and child angler/sportsman were based on the assumption that the intake for the adolescent will be approximately two-thirds that of the adult, and the intake for the child will be approximately one-third that of the adult (USEPA 2011a). Therefore, the adolescent and child fish ingestion rates used in the BHHRA are as follows:

- RME adolescent angler = 23.1 g/day
- CTE adolescent angler = 2.6 g/day
- RME child = 11.5 g/day
- CTE child = 1.3 g/day.

The uncertainty associated with the fish consumption rates is discussed in Section 7.

#### *4.3.6.2 Crab Ingestion Rate*

As explained above, fish and crab ingestion rates used in the BHHRA were developed as part of the LPRSA BHHRA (USEPA 2012a, 2012b). USEPA Region 2 evaluated the data collected for the Burger (2002) study in the Newark Bay Complex of New Jersey to estimate crab consumption. The Burger study reported a 50$^{th}$ percentile ingestion rate of 3.0 g/day and a 90$^{th}$ percentile ingestion rate of 20.9 g/day. As was assumed for fish, crab ingestion rates for the child and adolescent receptors were estimated assuming rates that are one-third and two-thirds of the adult ingestion rates, respectively. The crab ingestion dates used in this BHHRA are:

- RME adult crabber = 21 g/day
- CTE adult crabber = 3 g/day
- RME adolescent crabber = 14 g/day
- CTE adolescent crabber = 2 g/day
- RME child = 7 g/day
- CTE child = 1 g/day.

The uncertainty associated with the crab consumption rates is discussed in Section 7.

#### *4.3.6.3 Fraction Ingested for Fish and Crab*

The fraction ingested parameter (FI) represents the fraction of fish and crab consumed by the receptors that are from the NBSA. Although it is possible that anglers/sportsmen catch and consume fish and crab from rivers and other water bodies in the area, the risk assessment conservatively assumes that 100% of the catch is obtained from the NBSA for both the RME and CTE scenarios. The uncertainty associated with the assumption that all of the angler's catch comes from the NBSA is discussed in Section 7.

#### *4.3.6.4 Cooking Loss for Fish and Crab*

A cooking loss factor for exposure from fish ingestion accounts for the amount of chemical in fish tissue that is lost during cooking and thus is not consumed by the receptor. A cooking loss of 0% is assumed for the

RME scenario for all COPCs, a conservative approach that accounts for the potential scenario wherein individuals habitually consume cooking juices and pan drippings in addition to the cooked fish tissue. The assumption for the CTE fish ingestion scenario is that individuals discard the cooking juices and pan drippings, only consuming the cooked fish tissue. Therefore, chemical-specific cooking loss factors were developed for these scenarios for PCDD/Fs, PCBs, dieldrin, and several pesticides (the DDx isomers DDE, DDD, and DDT; alpha (cis)- and gamma (trans)-chlordane; cis- and trans-heptachlor epoxide; mirex; cis- and trans-nonachlor, and hexachlorobenzene).

The cooking loss values used in the CTE fish consumption scenario represent the 50$^{th}$ percentile of the current empirical cooking loss data sets for combined skin-on/skin-off tissues of various fish species by various cooking methods. The majority of these data are related to PCDD/Fs, PCBs, and organochlorine pesticides, and are summarized in USEPA's 2000 Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories (Volume 2, Appendix C) (USEPA 2000). In 2012, AECOM drafted a technical memorandum to update the cooking loss values recommended in the 2011 USEPA Region 2 Risk Analysis and Risk Characterization (RARC) Plan for the Lower Passaic River Study Area. For this effort, the authors summarized the updated cooking loss literature on fish consumption and cooking loss for PCDD/Fs, PCBs, and DDx chemical groups, calculating cooking loss for each study on a mass balance basis and developing summary statistics for each of these three chemical groups (AECOM 2012b).[5] Since then, Rawn et al. (2013) published more cooking loss data for PCDD/Fs and PCBs for several fish species for three common cooking methods.

For this BHHRA, cooking loss data sets for PCDD/Fs and PCBs were compiled from both Rawn et al. (2013) and the 2012 Draft AECOM Technical Memorandum. While the authors of the draft technical memorandum suggested that cooking loss data specific to DL-PCBs be included in the PCDD/F cooking loss data set (due to similar cooking loss magnitude and broader biochemical congruencies), USEPA concluded that the DL-PCB data set was too sparse to justify extending the idea of "dioxin-like" properties to the underlying physicochemical properties that moderate parameters like cooking loss. Therefore, the few DL-PCB cooking loss data points were included in the larger PCBs cooking loss data set.

For pesticides, cooking loss estimates were derived solely from the cooking loss data summarized for each respective chemical or chemical group in the 2000 USEPA Guidance. It should be noted that a cooking loss of 0% for all metals is assumed for both the RME and CTE scenarios per USEPA recommendation. This is because metals distribute in a manner different from that of organochlorine pesticides, tending to concentrate in liver, kidney, and/or muscle tissues, with little evidence of reduction from fillets after cooking

[5] Note: The 2012 Draft AECOM technical memo on cooking loss in fish was received by ToxStrategies in March/April 2012 for review and comment. ToxStrategies never received the final version that was submitted to USEPA July 5, 2012, although the cooking loss distribution analysis included in the 2017 LPRSA BHHRA document (Figure 7-2 of that document) is nearly identical to the analogous figure in the March 27, 2012, draft technical document.

(USEPA 2000). A cooking loss of 0% was also assumed for organics (benzaldehyde and pyridine) and PAHs, because the CL data are inadequate for developing values sufficient for risk assessment.

As with fish, the cooking loss factor for crab accounts for the amount of chemical in crab tissue that is lost during the cooking process and thus is not consumed by the receptor. Blue crabs are most often cooked whole by boiling or steaming (Sea Grant Marine Advisory Program 2006). For this reason, exposure to the chemicals in the whole crab, even the uneaten parts, may still occur if the liquid used to boil the crab is used in soups or other prepared dishes. Unlike the cooking loss data set for fish, however, the cooking loss literature for crab tissue is very sparse, with only a single relevant study (Zabik et al. 1992) reporting an approximately 20% reduction of PCBs from the tissue of steamed or boiled blue crabs (the species relevant to NBSA). The study found that about 80% of the PCBs is lost from the crab tissues in the cooking water. However, this data set (single study) is insufficient for use in the BHHRA. Therefore, it is assumed in this BHHRA that the cooking liquid is consumed along with the crabmeat, and thus, the CTE cooking loss factor for crab is assumed to be zero for all chemicals. Cooking loss factors for fish and crab assumed in the BHHRA are provided in Table 4-14.

### 4.3.7 Sediment and Surface Water Exposure Parameters

As shown in Figure 4-1 and Table 4-1, some activities that occur at the NBSA could result in direct contact with both surface water and sediment. Exposure parameters specific to the assessment of potential exposure to COPCs resulting from direct contact with sediment and surface water include incidental ingestion rates of sediment and surface water, body surface areas in contact with sediment and surface water, sediment-to-skin adherence factors, surface water exposure time, and sediment and surface water exposure frequencies. The following subsections discuss these exposure parameters.

#### *4.3.7.1 Incidental Ingestion of Sediment*

Studies on incidental ingestion of soil have been conducted, but similar data for sediment are lacking (USEPA 2011a). It is expected that some level of sediment removal will result in less hand-to-mouth loading than is the case with soil ingestion. In the BHHRA, the following assumptions are used:

- RME incidental ingestion of sediment occurs at a rate that is 50% of the recommended USEPA default values of 100 mg/day for adults and 200 mg/day for children. Thus, the BHHRA uses 50 mg/day for adults and adolescents and 100 mg/day for children in the RME scenarios that involve potential contact with sediment.
- For the CTE scenarios, the BHHRA assumes sediment ingestion rates that are 50% of the assumed RME rates; that is, 25 mg/day for adults and adolescents and 50 mg/day for children.

#### *4.3.7.2 Incidental Ingestion of Surface Water*

Data for incidental surface water ingestion during activities such as fishing, wading, and boating are generally lacking. USEPA has developed recommended default values for incidental ingestion of water during swimming (USEPA 2011a). For the BHHRA it is assumed that incidental ingestion of surface water by

the child and adolescent receptor during swimming occurs at the USEPA-derived mean rate for children (6–15 years old) of 0.05 L/hr. It is assumed that the adult rate during swimming is the USEPA mean rate of 0.021 L/hr for adults. These rates are used for both the RME and CTE swimmer scenarios. The incidental surface water ingestion rate for anglers/sportsmen, waders, and boaters is assumed to be half of what occurs during swimming, or 0.025 L/hr for children and adolescents and 0.011 L/hr for adults, for both the RME and CTE scenarios.

#### *4.3.7.3 Skin Surface Areas in Contact with Sediment and Surface Water*

The skin (dermal) surface area exposed to surface water and sediment is dependent on type of activity and receptor. Different activities are assumed to result in exposure of different body parts. Different receptors (adult, adolescent, or child) have different surface areas corresponding to body parts. The table below summarizes the assumptions for exposed skin surface area used in the BHHRA. The average of the mean values for male and female skin surface areas reported by USEPA (USEPA 2011a, 2014) are used. The same skin surface areas are used for the RME and CTE scenarios in the BHHRA. The BHHRA makes the following assumptions about exposed skin:

- Anglers/sportsman and waders are assumed to wear short-sleeved shirts and shorts (no shoes); therefore, the exposed skin surface is limited to the head (face), hands, forearms, lower legs, and feet.
- Adult boaters are assumed to wear shoes, and their exposure to surface water (due to splashing) is assumed to be limited to the hands, forearms, and face. Adolescent boaters are assumed to wear shorts or bathing suits and no shoes, so their exposure to surface water includes the lower legs and feet, as well as the hands, forearms, and face.
- During swimming by all age groups, the entire skin surface area is used for contact with surface water. Swimmers' dermal contact with sediment as they enter and leave the Bay is not likely to involve the entire body but would be similar to the exposure of a wader. Therefore, the exposed skin surface for sediment is assumed to be limited to the head, hands, forearms, lower legs, and feet.
- Workers are assumed to wear short-sleeved shirts, long pants, and shoes; therefore, for dermal contact with sediment, their exposed skin surface is limited to the head, hands, and forearms.

The specific input parameters for skin surface area contacting sediment or surface water are provided in Tables 4-2 through 4-11.

#### *4.3.7.4 Sediment-to-Skin Adherence Factors*

The adherence factor of 0.3 mg/cm$^2$ for adults is based on the geometric mean of the reed gatherer population from Exhibit 3-3 of RAGS Part E (USEPA 2004b) and is a weighted adherence factor based on hands, lower legs, forearms, and feet. The adherence factor based on reed gathering is a reasonable assumption for evaluating dermal exposure to NBSA sediment during recreational and worker activities. These activities all involve exposure of similar body parts, and reed gathering actions are reasonably comparable to those involved in the recreational and worker activities at the NBSA. The sediment-to-skin adherence factor for children and adolescent receptors is 0.2 mg/cm$^2$ based on the 50$^{th}$ percentile surface-

area-weighted soil adherence data for a child playing in wet soil (USEPA 2004b). These adherence values are applied to both the RME and CTE scenarios.

#### *4.3.7.5 Surface Water Exposure Time*

The exposure time, frequency, and duration of exposure to surface water at the NBSA are reasonably assumed to be relatively low, both currently and in the future, due to the deterrents to recreational use of the Bay. These deterrents include the presence of trash and debris, pathogenic contamination, ship traffic, and the general urban and industrial setting of the NBSA. They also limit the number of people who use the Bay in such a way as to be exposed to surface water. The angler/sportsman and wader exposure times used in the BHHRA are based on best professional judgment; CTE exposure times are assumed to be one-half of the RME exposure time. The swimmer exposure time is the national average for swimming, as reported in USEPA (1989), for both the RME and CTE scenarios. The RME exposure time to surface water for boaters is also based on professional judgment. CTE exposure time to surface water for boaters is assumed to be three-quarters of the RME exposure time. The surface water exposure time assumptions used in the BHHRA are provided in Tables 4-2 through 4-5, 4-7, 4-9, and 4-11.

#### *4.3.7.6 Sediment and Surface Water Exposure Frequencies*

Sediment and surface water exposure frequencies are based on site-specific conditions at the NBSA, including weather (i.e., cold months and frozen conditions limit exposure), type of recreational activity, and worker schedules. The exposure frequencies assumed in the BHHRA are provided in Tables 4-2 through 4-11. These frequencies are not expected to increase in the future. The bases for these assumptions are as follows.

<u>Angler/Sportsman</u>
For the RME scenario, adult and adolescent anglers are assumed to be exposed to sediment and surface water twice per week for 5.5 months per year of fishing (48 days/year) and twice per week for 3.5 months per year (30 days/year) of crabbing (Burger 2002). The CTE scenario for anglers assumes exposure frequencies that are one-half of the RME frequencies. Anglers are expected to contact surface water and sediment every day that they fish.

<u>Wader and Swimmer</u>
Adult and child receptors involved in wading and swimming in the Bay are assumed to be exposed to sediment and surface water one day per week for 3 months per year (June, July, and August) under the RME scenario (13 days/year). For the CTE scenario, one half of the RME exposure frequency is assumed (7 days/year). For wading and swimming, adolescents are assumed to have an RME sediment and surface water exposure frequency of 3 days per week for 3 months per year (39 days per year). Again, for the adolescent CTE scenario, one half of the RME exposure frequency is assumed (20 days/year).

<u>Boater</u>
For the boating scenario, the adult RME exposure frequency for surface water is assumed to be 7 days per week for 37 weeks per year (259 days/year). Adult boaters are assumed to have a CTE exposure frequency

to surface water of 3 days per week for 37 weeks per year (111 days/year). For sediment, the adult boater RME exposure frequency is assumed to be one day per month for 8.5 months per year (9 days/year). The adult CTE exposure frequency to sediment during boating is assumed to be one-half of the RME value (4 days/year).

Adolescents are assumed to be exposed to surface water during boating for 7 days per week for 14 weeks per year (98 days/year) under the RME scenario and 5 days per week for 14 weeks per year (70 days/year) under the CTE scenario. Adolescents are assumed to have an RME sediment exposure frequency during boating of 3 days per week for 3 months per year (39 days per year). For the adolescent CTE boating scenario, one half of the RME exposure frequency is assumed (20 days/year).

Worker

The adult worker is assumed to be exposed to sediment 1 day per week for 50 weeks per year (50 days per year) for the RME scenario and 1 day per week for 25 weeks per year (25 days) for the CTE scenario.

### 4.3.8 Exposure Durations

The exposure duration (ED) is the estimate of the total time (in years) that a receptor engages in a particular activity that could result in exposure. The ED assumptions for each of the four receptor populations (adult, adolescent, child, and worker) reflect differences in age span or type of activity (recreation vs. working). The same EDs are used for all recreational activities (angling, wading, swimming, boating) for a given receptor population.

Recreators (Angler/Sportsman, Swimmer, Wader, Boater)

The adult recreator (fishing, wading, swimming, and boating) is assumed to have an RME ED of 20 years (USEPA 2014). This is based on assuming a 26-year upper-bound residential tenure at a single location, minus 6 years as a non-adult (USEPA 2014). The CTE ED for the adult recreator is 9 years, based on the 50$^{th}$ percentile value for years living in current home (USEPA 2011a).

The adolescent recreator (fishing, wading, swimming, and boating) is assumed to have an RME ED of 12 years. This is the duration of the assumed adolescent age category (7 to <19 years old). The CTE ED (6 years) is assumed to be one-half of the RME value.

The child recreator (eating fish/crab, wading, and swimming) is assumed to have an RME ED of 6 years. This is the duration of the child age category (1 to <7 years old). The CTE ED (3 years) is assumed to be one-half of the RME value.

Workers

For the adult worker receptor population, the assumed ED is 25 years for the RME scenario, which is based on the 95$^{th}$ percentile for the number of years worked at the same location, as reported by the U.S. Bureau of Labor Statistics in 1990 (USEPA 1991b, 2014), and 7 years for the CTE, which is reported in the USEPA Exposure Factors Handbook (USEPA 2011a) as the median occupational tenure of the working population ages 16 and older (men and women).

### 4.3.9 Body Weights

Receptor body weights are taken from USEPA guidance (USEPA 2011a, 2014) and represent the averages for males and females in the applicable age ranges (i.e., 1 to <7 years for child, 7 to <19 years for adolescent, and adult). A body weight of 80 kg is used for adults (USEPA 2014). Body weights for young children and adolescent age groups were derived by averaging the mean body-weight estimates for males and females by year of age from the National Health and Nutrition Examination Survey, as summarized in Table 8-24 of the USEPA Exposure Factors Handbook (USEPA 2011a). The mean body weight assumed in the BHHRA is 17 kg for the 1- to <7-year-old child and 52 kg for the 7- to <19-year-old adolescent. The same body weights are used for RME and CTE scenarios.

### 4.3.10 Chemical-Specific Exposure Parameters

This section presents assumptions used in the BHHRA for exposure parameters that are chemical-specific, including dermal absorption fractions, oral absorption adjustment factors, and factors related to dermal permeability of chemicals in water. Chemical-specific cooking loss factors were presented and discussed in Section 4.3.6.4 above.

### 4.3.11 Dermal Absorption Fractions

The dermal absorption fraction (DAF) accounts for absorption of chemicals through the skin from dermal contact with sediment. The DAFs for COPCs were compiled from RAGS Part E (USEPA 2004b), consistent with the USEPA Regional Screening Levels (USEPA 2019a) and are presented in Table 4-12. Default DAFs provided in USEPA (2004b) have been used in the BHHRA. The uncertainty associated with using default DAFs is discussed in the uncertainty evaluation (Section 7).

### 4.3.12 Oral Absorption Adjustment Factors

Oral relative bioavailability (RBA) is the ratio between the estimated human absorption factor of a chemical (for the specific medium and route of exposure) and the estimated absorption factor for the laboratory study from which the dose-response value was derived (also referred to as absorption adjustment factor). In the BHHRA, as recommended by USEPA (USEPA 1989, 2019a), this factor is assumed to be 100% (RBA = 1) for all chemicals except arsenic. The value for arsenic is assumed to be 0.6 (60%), as derived by USEPA for soils based on a review of over 100 arsenic RBA estimates (USEPA 2012c). The oral RBAs used in the BHHRA are also listed in Table 4-12. The uncertainty associated with using a default RBA for arsenic in soil to estimate exposure to arsenic in sediment is discussed in the uncertainty evaluation (Section 7).

#### *4.3.12.1 Dermal Water Parameters*

Estimating chemical intake through dermal contact with surface water is done in the BHHRA by a method discussed by USEPA (USEPA 1989, 2004b). The approach uses a dermal permeability coefficient (Kp), which reflects the rate of migration of the chemical through the skin. The method involves a two-compartment distributed model that describes absorption of chemicals in water through the skin as a

function of both the event duration and the thickness of the stratum corneum. This approach assumes that the action of chemicals dissolved in water is consistent with Fick's second law. Fick's law describes the absorption process whereby a chemical is only in the stratum corneum, as well as absorption once steady state is reached. The model assumes that absorption continues after the exposure has ceased; therefore, the final absorbed dose (DAevent) is calculated as the total dose dissolved in the skin at the end of the exposure time (USEPA 1989, 2004b). This method uses default as well as chemical-specific dermal water parameters, including:

- A dermal permeability coefficient, described above (Kp, cm/hr)
- Fraction absorbed, which accounts for loss of chemical due to desquamation of the outer skin layer (FA, dimensionless)
- The ratio of the permeability coefficient of a chemical through the stratum corneum relative to its permeability coefficient across the viable epidermis (B, dimensionless)
- Lag time per event (τ, or tau event, hours/event)
- Time to reach steady state (t*, hours).

Chemicals are classified by USEPA (2004b, 2019a) as being within or outside the effective prediction domain (EPD). In general, chemicals with very high or very low octanol/water partition coefficient (Kow) values are outside the EPD. The EPD is determined using a chemical's molecular weight and Kow. The EPD is the region on an X/Y graph that represents 95% statistical confidence levels of a regression equation to reliably estimate Kp, Kp is estimated and the dermal exposure to water route is evaluated only if a chemical is within the EPD (USEPA 2004b, 2019a).

These parameters are presented in Table 4-13 for the COPCs and are from USEPA guidance (USEPA 2004b), consistent with the USEPA RSLs (USEPA 2019a).

## 4.4 Exposure-Point Concentrations

Exposure-point concentrations (EPCs) are estimates of the concentrations of COPCs in environmental media at the locations where humans may have contact with these media. EPCs are used to determine the magnitude of potential human exposure, as described in Section 4.3. The methods used to calculate EPCs are presented in the rest of this section. For this BHHRA, EPCs were derived using measurements of COPC concentrations in accessible surface sediment samples (Section 4.4.3), surface water samples (Section 4.4.4), and fish/crab tissue (Section 4.4.5).

### 4.4.1 Calculation of Exposure-Point Concentrations

In each exposure medium, the EPC for each COPC is defined as the 95% upper confidence limit (UCL) on the mean concentration. The 95% UCL represents a reasonable upper bound on the arithmetic average concentration that is contacted over the exposure period, accounting for uncertainty in estimating the true average concentration at an exposure point; it is used according to USEPA guidance (USEPA RAGS-A Guidance [1989]). In the event the 95% UCL is greater than the maximum reported concentration, the

maximum concentration was used as the EPC (this case occurred only once, for PCB-189 in accessible surface sediment).

#### *4.4.1.1 Treatment of Duplicate Values*

During the process of calculating EPCs, field duplicates were averaged together with their parent samples. Specifically, if both parent and duplicate samples were detected, or both were non-detects, then the two values were averaged to yield a single combined value (which was assigned qualifier J if both were detects, and qualifier U if both were non-detects). If only one of the parent or duplicate values was detected and the other was non-detect, then the combined value was assigned to be the detected value, and was assigned the qualifier of the detected value (if any). This approach stands in contrast to the approach used while identifying COPCs, wherein duplicate values were treated as independent samples.

#### 4.4.1.2 *Use of ProUCL software*

USEPA's ProUCL software (version 5.1) was used to calculate 95% UCLs. ProUCL takes input consisting of measured values for each COPC in a given medium, with corresponding numerical flags indicating whether each measured value was detected (flag value 1) or a non-detect (flag value 0). Values with qualifier U were assigned detection flag 0; other values (including those with qualifier J) were assigned detection flag 1. (Any values with qualifier R, indicating rejected data, had already been removed from consideration at the stage of identifying COPCs; see section 3.1.) Any non-detect values were entered into ProUCL as originally reported in the data. ProUCL automatically identifies the appropriate statistical methods to handle non-detects while estimating EPCs.

ProUCL calculates multiple estimates of the 95% UCL, using multiple parametric distributional assumptions and non-parametric methods, and compares these estimates using goodness-of-fit measures. Ultimately, ProUCL recommends one or more 95% UCL estimates. In some cases, ProUCL's recommended UCL estimate was inappropriate. These included H-UCLs (UCLs based on Land's H-statistic); GROS Adjusted Gamma UCLs (GROS stands for Gamma Regression on Order Statistics); GROS Approximate Gamma UCLs; and any UCL that was not a 95% UCL (e.g., ProUCL occasionally recommends a 97.5% UCL or a 99% UCL, rather than a 95% UCL).

H-UCLs are computed by ProUCL only for reasons of historical comparison, and the ProUCL Technical Guidance explicitly states that the H-UCL should not be used (USEPA 2015a). However, this aspect of the technical guidance has not been implemented in the ProUCL software; the software still occasionally selects the H-UCL as its recommended UCL.

GROS Adjusted Gamma and GROS Approximate Gamma UCLs were considered inappropriate for a different reason. The GROS method is a method for imputing values for non-detects, based on a gamma distribution estimated from the detected values. However, when the data set of detected observations is highly skewed, the GROS method does not perform well, and tends to impute negative values for non-detected values. Because environmental concentration data cannot be negative, ProUCL automatically substitutes any negative imputed values with a constant value of 0.01 (USEPA 2015a). The 0.01 substitution

value is hard-coded into the software and cannot be changed. A value of 0.01 was often on an inappropriate scale for the data sets under consideration in this BHHRA. As a hypothetical example, the maximum reporting limit for a COPC in fish fillet may have only been $1\times10^{-6}$ mg/kg; in this case, substituting non-detect values with 0.01 mg/kg is obviously inappropriate.

ProUCL Technical Guidance (USEPA 2015a) states that GROS should not be used for data sets where the detected observations are highly skewed. The Technical Guidance defines "highly skewed" data as data where the estimated value of one parameter of the gamma distribution, *k*, is less than 1. However, in some cases, data sets did not meet ProUCL's criterion for "highly skewed," and a GROS UCL was recommended, but the GROS UCL was still substantially higher than the general scale of the data, suggesting that one or more substitutions of 0.01 had been made. For example, for 1,2,3,4,7,8-HxCDD in summer flounder fillet samples, a GROS UCL was one of ProUCL's recommended UCLs. For this data set, the parameter *k* was estimated at 3.62, well above the cutoff value of 1 in the ProUCL Technical Guidance, suggesting that the detected data were not "highly skewed." However, the GROS UCL exceeded the maximum detected value by several orders of magnitude: the maximum detected value was $1.64\times10^{-7}$ mg/kg, but the GROS UCL was $7\times10^{-3}$ mg/kg. Situations like this one caused GROS UCLs to be eliminated from consideration as EPC estimates.

We applied the following algorithm to choose a 95% UCL estimate from those computed by ProUCL.

1. The algorithm selected the maximum ProUCL-recommended UCL that was *not* an H-UCL, a GROS UCL, or a 97.5% or 99% UCL.
2. If no recommended UCL satisfied the conditions in (1), then the algorithm defaulted to selecting the 95% Chebyshev UCL (following guidance in the ProUCL Technical Guidance, where UCLs based on the Chebyshev inequality are considered reasonably stable and conservative estimates for skewed data sets).

For data sets where there were not enough distinct detected values for ProUCL to estimate any UCL, the maximum reported concentration was used as the EPC (whether it was detected or non-detected).

#### 4.4.2 Exposure Areas

Exposure areas are discrete areas of the site over which exposure is expected to occur throughout the duration of exposure. For the BHHRA, exposure to accessible surface sediment, surface water (depth up to 3 feet), and fish or crab tissue were assumed to occur over the entirety of Newark Bay (i.e., sitewide [or Bay-wide]).

#### 4.4.3 EPCs for Sediment

EPCs were calculated for COPCs in accessible surface sediment samples on a site-wide basis (see Table 4-15 [RAGS Part D Table 3.1]).

### 4.4.4 EPCs for Surface Water

EPCs were calculated for COPCs in surface-water samples (near-surface only; i.e., depth up to approximately 3 feet) on a site-wide basis (see Table 4-16 [RAGS Part D Table 3.2]).

### 4.4.5 EPCs for Fish Tissue

For fish fillets, EPCs were calculated on a site-wide basis (see Table 4-17 [RAGS Part D Table 3.3]). If a chemical was a COPC in any fish species or crab tissue, an EPC was computed in all fish species and crab tissues. Ultimately, species-specific EPCs were averaged across all five fish species as described below to yield an EPC for a mixed-fish diet, assuming that an angler eats approximately equal quantities of fish fillets from each species over the period of exposure.

For each COPC, the mixed-fish diet EPC was calculated by averaging species-specific EPCs, rather than calculating an EPC for all fish fillet data pooled together, because there was not necessarily an equal number of measured values in each fish species. An EPC calculated from pooled fish fillet data would therefore reflect the relative availability of measured values in each fish species, not a diet composed of equal parts of each fish species. For this reason, the mixed-fish diet EPC was calculated by first calculating EPCs for each species separately, and then averaging the species-specific EPCs (see Table 4-18 [RAGS Part D Table 3.4]).

### 4.4.6 EPCs for Crab Tissue

EPCs for crab tissue were also calculated on a site-wide basis (see Table 4-19 [RAGS Part D Table 3.5]). If a chemical was a COPC in any fish species or crab tissue, an EPC was computed in all fish species and crab tissues. EPCs are computed for the combined muscle/hepatopancreas tissue (assuming 74% muscle and 26% hepatopancreas, by mass (see Attachment A-6 to Appendix A), as well as for muscle tissue only and for hepatopancreas tissue only. Note that for combined muscle/hepatopancreas tissue, the combined COPC concentrations were calculated for each individual sample first, and then EPCs were computed using ProUCL as 95% UCLs based on this data set of combined concentrations. (This stands in contrast to the other possible approach, which would be to calculate muscle EPC and hepatopancreas EPC separately, and then take a weighted average of the two tissue-specific EPCs.)

## 5. Toxicity Assessment

The intent of the toxicity (and dose-response) assessment is to determine the nature of adverse health effects that may occur with exposure to a certain chemical, and to identify the relationship between the dose of a chemical and the possibility and extent of a potential adverse effect (or response) (USEPA 1989). Cancer risk and noncancer hazard can be estimated by incorporating the outcome of the toxicity assessment with information on the magnitude of potential exposure (developed in the exposure assessment) to provide an estimate of potential risk (provided in the risk characterization).

USEPA designates potential adverse effects as carcinogenic or noncarcinogenic (i.e., effects other than cancer). Dose-response associations are generally defined by USEPA for oral and inhalation exposures. Due to the lack of toxicity data for the dermal exposure route, oral toxicity values adjusted for absorption differences are typically used to evaluate dermal exposures (USEPA 2004b).

Potential noncancer health effects, likely caused by a nonlinear mode of action, are evaluated using oral reference doses (RfDs) (USEPA 2019b). Noncancer toxicity values are derived assuming that various toxic consequences (e.g., renal effects) have threshold concentrations. RfDs are estimates (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (USEPA 2019b). For characterization of potential noncarcinogenic effects, exposures are classified as chronic (i.e., 7 years to a lifetime) or subchronic (i.e., <7 years) (USEPA 2019c). In USEPA's soil screening-level guidance (USEPA 2002a), the Science Advisory Board indicates that, although a conservative assumption, a child scenario with 6 years of exposure can be paired with a chronic RfD. RfDs are expressed in milligrams of a chemical per kilogram of body weight per day (mg/kg-day).

Potential cancer effects are evaluated primarily using oral cancer slope factors (CSFs), which are expressed as risk per mg/kg-day. In addition, USEPA has developed weight-of-evidence characterizations for determining human carcinogenicity (USEPA 2019b).

Note that USEPA also derives inhalation toxicity factors, but these are not discussed in the BHHRA, as inhalation exposures due to volatilization of chemicals from sediment and surface water were evaluated separately and their impacts were found to be negligible (see Section 4.1, Appendix C-1 and C-2). For sediment, all estimated cancer risks were below $10^{-6}$ (maximum of $1.4\times10^{-7}$) and all noncancer hazard quotients were below 1 (maximum of 0.16). For surface water, volatilization of COPCs was evaluated using a tiered approach. For those chemicals for which the Tier 1 air concentration exceeded USEPA's residential air RSLs, USEPA's AERSCREEN model was applied, and the Tier 2 concentrations for these COPCs were below the residential RSLs by at least an order of magnitude. Additional information can be found in Section 4.1, and the complete documentation can be found in Appendix C-1 (sediment) and Appendix C-2 (surface water).

The toxicity assessment is presented in the subsections below. Section 5.1 discusses the sources of toxicity data used in the BHHRA. Section 5.2 discusses the noncarcinogenic toxicity factors, and Section 5.3 discusses the carcinogenic toxicity values. Section 5.4 discusses the gastrointestinal absorption values used

to adjust oral toxicity factors to evaluate the dermal pathway. Section 5.5 discusses unique toxicity evaluations for dioxins and furans, PCBs, PAHs, arsenic, lead, and mercury.

### 5.1 Sources of Toxicity Data

Dose-response relationships forming the basis of toxicity factors, particularly older values, are typically derived from laboratory animal experiments that include a control and testing at varying dose levels. The data from these studies are used to determine the critical effects and the associated dose levels in the development of the toxicity values. This information is then used by USEPA to develop the RfD or CSF that is then externally reviewed and made available through mechanisms such as IRIS, PPRTVs, etc. Further, the studies are performed under prescribed conditions intended to decrease the impact of confounding factors. The relatively high doses used in animal studies are then extrapolated to lower concentrations relevant to humans using uncertainty factors (UFs) and toxicological models.

Toxicity factors used in BHHRA were selected according to USEPA's toxicity factor hierarchy (USEPA 2003a). The first choice for toxicity values is USEPA's Integrated Risk Information System (IRIS), a toxicity database available online. The IRIS program resides within the National Center for Environmental Assessment's (NCEA's) Office of Research and Development (ORD) (USEPA 2019b). Using IRIS guidance, USEPA reviews and assesses toxicological studies on health effects potentially relevant to humans resulting from exposure to various substances. The current IRIS process (NRC 2014) involves selection of the substance for evaluation, problem formulation, systematic review and comprehensive literature search, draft assessment for agency and public review, peer review, and release of the final assessment (https://www.epa.gov/iris/basic-information-about-integrated-risk-information-system#process). USEPA's toxicity factor hierarchy (USEPA 2003a) is as follows:

- Tier 1 — USEPA's IRIS (USEPA 2019b)
- Tier 2 — USEPA's Superfund Health Risk Technical Support Center (STSC) Provisional Peer Reviewed Toxicity Values (PPRTV) (USEPA 2019d)
- Tier 3 — Other toxicity values, such as PPRTV screening values (USEPA 2019d), California Environmental Protection Agency (CalEPA) values, Agency for Toxic Substances and Disease Registry (ATSDR) Minimal Risk Levels (MRLs) (ATSDR 2019), and Health Effects Assessment Summary Tables (HEAST) toxicity values (USEPA 1997a), with preference given to sources based on approaches similar to those used for Tiers 1 and 2, peer-reviewed values, publicly available values, more recent values, and toxicity factors that are transparent in their development (USEPA 2003a).

The sediment and surface water inhalation pathways were evaluated and excluded from the BHHRA, because the risks and hazards from these pathways are considered negligible (Appendices C-1 and C-2), and inhalation toxicity factors are not relevant in the risk assessment. Therefore, oral RfDs and oral cancer CSFs were used, as well as oral toxicity factors adjusted for dermal absorption. Noncancer toxicity values used in this risk assessment are shown in Table 5-1 (RAGS Part D Table 5.1); cancer toxicity factors are shown in Table 5-2 (RAGS Part D Table 6.1). As depicted in Tables 5-1 and 5-2, most of the toxicity factors used in this assessment are Tier 1 values from the USEPA IRIS database (2019b); PPRTVs (USEPA 2019d) are included as Tier 2 values; and Tier 3 values include PPRTV screening toxicity values (USEPA

2019d); values from the CalEPA Toxicity Criteria Database (CalEPA 2019); and values from NJDEP (2009), ATSDR MRLs (2019), and HEAST (USEPA 1997a).

PPRTVs (Tier 2) are used for the following COPCs and toxicity values:

- Aluminum RfD (USEPA 2006a)
- Benzaldehyde CSF (USEPA 2015b)
- Cobalt RfD (USEPA 2008)
- Iron RfD (USEPA 2006b)
- PHC as gasoline RfD (USEPA 2009b)
- TPH (C9-C40) RfD (USEPA 2009b).

If toxicity factors are not available from IRIS (Tier 1 source) or the PPRTVs (Tier 2), a Tier 3 source is applicable. PPRTV screening values are derived when sufficient information is not available to derive a PPRTV, but USEPA's Superfund Health Risk Technical Support Center (STSC) has determined that adequate information is available that may be of limited utility to risk assessors. In these cases, the STSC derives a screening value and compiles available data in an appendix (USEPA 2012d, 2017e, 2017f). Therefore, these screening toxicity factors are treated as Tier 3 values. Previously, HEAST was published yearly by USEPA in hard-copy form; the most recent update was in 1997. Therefore, some of these values may be outdated. As discussed in Section 7.3.6, the only HEAST toxicity factor used in this risk assessment is the CSF for dioxin-like compounds (DLCs).

Per USEPA (2003a), Tier 3 toxicity values were chosen by giving priority to those that are newer, transparent, peer reviewed, and publicly available. Tier 3 toxicity values used in this assessment are as follows:

- 2,4'-DDD — PPRTV screening chronic RfD for 4,4'-DDD (4,4'-DDD used as a toxicity surrogate) (USEPA 2017e)
- 2,4'-DDE — PPRTV screening chronic RfD for 4,4'-DDE (4,4'-DDE used as a toxicity surrogate) (USEPA 2017f)
- 4,4'-DDD — PPRTV screening chronic RfD (USEPA 2017e)
- 4,4'-DDE — PPRTV screening chronic RfD (USEPA 2017f)
- Chloroform — CalEPA CSF (listed by CalEPA as CalEPA 2011, CARB 1990)
    - Note that the CalEPA toxicity criteria database lists an oral CSF of 3.1E-02 mg/kg-day$^{-1}$ (used in this BHHRA) and a less conservative inhalation CSF of 1.9E-02 mg/kg-day$^{-1}$. The documentation cited by CalEPA refers to derivation of only the less conservative inhalation CSF value (CalEPA 2011) and ranges of slope factors (CARB 1990). Further, chloroform is unique, in that the IRIS file states that the RfD is also protective of cancer (USEPA 2019b).
- Chromium (VI) — NJDEP CSF (NJDEP 2009)
- Copper — ATSDR intermediate MRL (ATSDR 2004), with a UF of 10 applied (USEPA and NJDEP 2019)
- Mirex — CalEPA CSF (CalEPA 1992)

- Organic arsenic — ATSDR chronic MRL (used as the RfD) (ATSDR 2007) (see Section 5.5.4 for further information)
- PCDD/Fs and DL-PCBs — HEAST (USEPA 1997a) CSF for TCDD with TEF applied (see Section 5.5.1)
- Thallium — PPRTV screening value chronic RfD (USEPA 2012e) (see Section 5.2 for additional information).

Where toxicity factors were not available from any of the recommended sources, a toxicity factor for a structurally similar compound was assigned as a surrogate (also see Tables 5-1 and 5-2). Based on input from USEPA Region 2 (USEPA 2018a, 2018d), USEPA's Superfund Health Risk Technical Support Center (STSC, USEPA 2015c through 2015g), as well as surrogates based on structural and/or toxicological similarities, surrogates were chosen for the following COPCs in the BHHRA.

| COPC* | RfD Surrogate | CSF Surrogate |
|---|---|---|
| PCDD/Fs and DL-PCBs | Value for 2,3,7,8-TCDD with TEF applied | Value for 2,3,7,8-TCDD with TEF applied |
| Total Non-DL PCBs (RME) | Aroclor 1254 | Polychlorinated Biphenyls (high risk and persistence, upper-bound slope factor) |
| Total Non-DL PCBs (CTE) | Aroclor 1254 | Polychlorinated Biphenyls (high risk and persistence, central-estimate slope factor) |
| Benz(a)anthracene | -- | Value for Benzo(a)pyrene with RPF applied |
| Benzo(b)fluoranthene | -- | Value for Benzo(a)pyrene with RPF applied |
| Benzo(k)fluoranthene | -- | Value for Benzo(a)pyrene with RPF applied |
| Chrysene | -- | Value for Benzo(a)pyrene with RPF applied |
| Dibenz(a,h)anthracene | -- | Value for Benzo(a)pyrene with RPF applied |
| Indeno(1,2,3-c,d)-pyrene | -- | Value for Benzo(a)pyrene with RPF applied |
| 2,4'-DDD | 4,4'-DDD | 4,4'-DDD |
| 2,4'-DDE | 4,4'-DDE | 4,4'-DDE |
| 2,4'-DDT | 4,4'-DDT | 4,4'-DDT |
| Chlordane, alpha (cis) | Chlordane | Chlordane |
| Chlordane, gamma (trans) | Chlordane | Chlordane |
| Heptachlor epoxide, trans- | Heptachlor epoxide | Heptachlor epoxide |

| COPC* | RfD Surrogate | CSF Surrogate |
|---|---|---|
| Nonachlor, cis- | Value for chlordane with RPF applied | Chlordane |
| Nonachlor, trans- | Value for chlordane with RPF applied | Chlordane |
| Oxychlordane | Value for chlordane with RPF applied | Chlordane |
| PHC as gasoline | Total Petroleum Hydrocarbons (Aromatic Low) | Total Petroleum Hydrocarbons (Aromatic Low) |
| TPH (C9-C40) | Total Petroleum Hydrocarbons (Aromatic Medium) | Total Petroleum Hydrocarbons (Aromatic Medium) |
| Arsenic, organic | Dimethylarsinic acid | Dimethylarsinic acid |
| Mercury | Mercuric Chloride | Mercuric Chloride |
| Nickel | Nickel Soluble Salts | Nickel Soluble Salts |
| Thallium | Thallium Soluble Salts | Thallium Soluble Salts |
| Titanium | Titanium Tetrachloride | Titanium Tetrachloride |

*Surrogate list above applies to toxicity factors for COPCs evaluated quantitatively in the BHHRA. Additional surrogates were applied to the screening levels used for COPC selection (Appendix B).

RPF — relative potency factor

Note that if a relevant surrogate is recommended in a STSC memorandum provided by USEPA Region 2 (USEPA 2015c through 2015g), that memorandum is cited.

### 5.2 Noncarcinogenic Toxicity Assessment

Chemicals associated with noncarcinogenic health effects are assumed to have a threshold (i.e., a level above which an adverse effect may occur, or a level below which no adverse effect is observed). The no-observed-adverse-effect level (NOAEL) is an estimate of the threshold dose. The minimum dose at which an adverse effect has been reported is called the lowest-observed-adverse-effect level (LOAEL). If available, the NOAEL (otherwise, the LOAEL) is employed as the point of departure (POD) for predicting a threshold level in humans based on extrapolations from experimental information. RfDs for chronic exposure to chemicals with noncancer effects have been estimated by USEPA by modifying the NOAEL or the LOAEL with UFs (1997a, 2019b).

More recently, USEPA has employed benchmark dose (BMD) methods to designate the POD for an adverse effect (benchmark response) from experimental studies (USEPA 2012f). The BMD method is a more quantitative option for the initial step in the dose-response assessment than the NOAEL/LOAEL approach. In deriving the BMD, response data are initially modeled within the range of experimental observations, then modeling is used to predict a value below the experimental range. In BMD modeling, the POD is the BMD lower confidence level (BMDL), which is the lower 95% bound on the dose that elicits the adverse effect, usually 10% higher than the control response. Uncertainty inherent in a given experiment is

considered via use of the lower bound, which also ensures with 95% confidence that the target benchmark response is not surpassed. The RfD is then derived by applying UFs to the BMDL.

RfDs are derived using the critical (most sensitive) adverse effect in the study, assuming that, if the most sensitive effect does not occur, no other potential toxic effects would occur. USEPA assumes that humans are at least as sensitive to a substance as the most sensitive laboratory species. To account for uncertainties inherent in the relationship between dose and response, the BMDL, NOAEL, or LOAEL is modified by UFs of 1, 3, and/or 10 (USEPA 2002b). UFs are applied to extrapolate from a subchronic to a chronic exposure, extrapolate from a LOAEL to a NOAEL, considering sensitive subpopulations, and using an animal study to derive a human toxicity factor. In addition, a modifying factor (MF) may be used to cover uncertainties in the database or study or that were not considered by other UFs (USEPA 2002b). For the COPCs evaluated in this BHHRA, total UFs range from 1 to 3,000, which is the maximum total UF recommended by USEPA (2002b). The resulting RfDs are considered to be health-protective. Specifically, the RfD represents a level of daily exposure for a lifetime that is likely to be without an appreciable risk of deleterious effects. The smaller the RfD value, the lower is the assumed threshold for noncancer effects; therefore, the compound is considered more toxic.

Table 5-1 provides the noncancer toxicity information for COPCs for the oral and dermal exposure routes. For the applicable COPCs, Table 5-1 lists the Chemical Abstracts Service (CAS) number, oral RfD, TEF for dioxin-like compounds, oral absorption efficiency for the dermal pathway, absorbed RfD for dermal, primary target organ(s), modifying/uncertainty factors, source, date, toxicity factor tier, surrogate, CAS for surrogate, and rationale/reference for surrogate. Adjustments for dermal absorption are discussed in Section 5.4.

PPRTV screening values are used as the RfDs for several chemicals, as discussed below. These provisional screening values are included in the appendices of relevant chemical-specific PPRTV documents. PPRTV screening values are less certain than standard PPRTVs but use current USEPA methodology in their derivation and undergo external peer review. However, these RfDs may be of limited utility to risk assessors as there is considerable uncertainty associated with these values (USEPA 2019a, 2019d). Any unacceptable hazards associated with these chemicals are discussed in the uncertainty evaluation to inform risk management decisions (Section 7).

The thallium RfD is a PPRTV screening value (USEPA 2012e). A standard PPRTV RfD was not derived due to the poor quality of the database. However, data are available that may be of limited use. Therefore, the Superfund Health Risk Technical Support Center compiled the available information and derived a PPRTV screening RfD. While the screening RfD was used in this risk assessment, it is noted that the thallium hazard quotients (HQs) are particularly uncertain considering the screening RfD. The RfD is based on a subchronic gavage study in rats using thallium soluble salts. The toxicity endpoint is atrophy of hair follicles, because hair follicle atrophy is consistent with episodes of thallium poisoning in humans (USEPA 2012e). However, hair follicle atrophy is not necessarily a toxic effect, which adds uncertainty to the thallium HQ.

The 4,4'-DDD RfD (also used as a surrogate for 2,4'-DDD) is a PPRTV screening value. A standard PPRTV RfD was not derived for 4,4'-DDD due to inadequate data. The existing chronic studies have issues regarding purity of the substance, inadequate number of doses and study animals, and excess mortalities in

the study animals. Due to the lack of toxicity data for 4,4'-DDD, USEPA used 4,4'-DDT as a surrogate to derive a PPRTV for 4,4'-DDD (USEPA 2017e). The screening PPRTV is based on a dietary study in rats (Laug et al. 1950, as cited in USEPA 2017e); the same study used to derive the IRIS oral RfD and ATSDR intermediate MRL for DDT. Rats were administered technical-grade DDT dissolved in corn oil in the diet for 15–27 weeks. The critical effect was liver lesions in males and females. The NOAEL (1 ppm [0.05 mg/kg-day]) was selected as the point of departure (POD); this value was converted to a human equivalent dose (HED) of 0.01 mg/kg-day using a dosimetric adjustment factor of 0.27. The resulting HED was divided by an uncertainty factor (UF) of 300 (interspecies UF of 3, intraspecies variability value of 10, database deficiency value of 10), resulting in a PPRTV screening chronic RfD of 3E-05 mg/kg-day (USEPA 2017e).

Similar to 4,4'-DDD above, the 4,4'-DDE RfD (also used as a surrogate for 2,4'-DDE) is a PPRTV screening value. A standard PPRTV RfD was not derived for 4,4'-DDD due to inadequate data. The existing chronic studies have issues regarding adjustment of doses during the studies, excessive long recovery period following exposure, and LOAELs close to mortality levels in some cases (USEPA 2017f). The screening PPRTV is based on a study in rats (Yamasaki et al. 2009, as cited in USEPA 2017f), whereby adult males exposed to DDE during gestation and lactation demonstrated significantly increased relative liver weights. The LOAEL was 5 mg/kg/day; BMD modeling was performed, and the resulting POD (HED) of 1 mg/kg-day was divided by a UF of 3,000 (interspecies UF of 3, intraspecies variability value of 10, LOAEL-to-NOAEL UF of 10, database deficiency value of 10), resulting in a PPRTV screening chronic RfD of 3E-04 mg/kg-day (USEPA 2017f).

Per USEPA and NJDEP (2019), USEPA requested additional information from the Superfund Toxicity Workgroup regarding the copper toxicity value. While an oral RfD is available in HEAST (USEPA 1997a), ATSDR has developed an intermediate MRL for copper of 0.01 mg/kg-day. The intermediate MRL is based on a 2003 study in humans who ingested copper sulfate in drinking water for 2 months. The value is based on a NOAEL of 0.042 mg/kg-day and application of a UF of 3 for human variability. The critical effect is GI effects (ATSDR 2004). The Tier 3 toxicity value hierarchy provided in USEPA (2013a) lists ATSDR MRLs before HEAST values. For this reason, as well as the fact that the ATSDR intermediate MRL for copper is based on a more recent study than that used for the HEAST (USEPA 1997a) value, the Superfund Toxicity Workgroup recommends use of the ATSDR intermediate MRL as the chronic RfD for copper, with an additional UF between 3 and 10 applied. As the STSC would need to conduct a thorough review of the toxicity data to assign the particular UF, which would take a substantial amount of time, USEPA Region 2 recommends application of a UF of 10. If the noncancer HI is exceeded, USEPA recommends a discussion of the impacts of using a factor of 3 as the UF in the uncertainty evaluation (Section 7). Therefore, USEPA's recommended copper RfD of 0.001 mg/kg-day (ATSDR Intermediate MRL/10) is used in the BHHRA (USEPA and NJDEP 2019), and additional discussion is provided in the uncertainty evaluation (Section 7).

The risks and hazards for COPCs with Tier 3 toxicity values are discussed in the uncertainty evaluation (Section 7).

### 5.3 Carcinogenic Toxicity Assessment

USEPA has issued revised risk assessment guidelines for carcinogens (USEPA 2005b), which replace the previous version (USEPA 1986b). As shown in Table 5-2, many of the COPCs still follow the 1986 classification system; the previous classification system is used until a chemical is reassessed in the IRIS program in accordance with the 2005 Cancer Guidelines. Weight-of-evidence information from animal and epidemiologic studies was used to develop the 1986 classification system:

- Group A — Human Carcinogen (sufficient evidence of carcinogenicity in humans)
- Group B — Probable Human Carcinogen (B1, limited evidence of carcinogenicity in humans; B2, sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans)
- Group C — Possible Human Carcinogen (limited evidence of carcinogenicity in animals and inadequate or lack of human data)
- Group D — Not Classifiable as to Human Carcinogenicity (inadequate or no evidence)
- Group E — Evidence of Noncarcinogenicity for Humans (no evidence of carcinogenicity in adequate studies).

In the 1986 guidance, USEPA assumed that a specific level of cancer risk is associated with every dose (USEPA 1986b). Mathematical models have been developed by USEPA that extrapolate dose-responses occurring at relatively high doses (used in animal studies) to the lower doses typically experienced by humans. These models assume no threshold for carcinogenic effects and use available animal and/or human data to estimate potency values (CSFs). CSFs are expressed in risk per mg/kg-day (or mg/kg-day$^{-1}$); therefore, the higher the CSF, the greater the potential for carcinogenicity.

USEPA's 2005 guidance focuses on evaluating all available information and incorporating mode-of-action (MOA) data (USEPA 2005b). A default, linear low-dose extrapolation may be used if data are lacking. MOA is a series of key processes and events, beginning with interaction of a compound with a cell, proceeding through anatomical and operational changes, and culminating in the development of cancer. MOAs that are expected to be mutagenic are evaluated using linear extrapolation. Other MOAs may be evaluated with either linear or nonlinear methods following careful review of available information per the 2005 guidance. USEPA's 2005 guidance details a weight-of-evidence description instead of the 1986 classification system. USEPA (2005b) includes the following descriptors along with the weight-of-evidence discussion:

- Carcinogenic to humans — indicates strong evidence of human carcinogenicity
- Likely to be carcinogenic to humans — used when the weight of evidence demonstrates carcinogenic potential for humans
- Suggestive evidence of carcinogenic potential — appropriate when the weight of evidence suggests carcinogenicity; a concern for potential carcinogenicity in humans is raised, but the data are insufficient for a substantial conclusion
- Inadequate information to assess carcinogenic potential — used when available data are not adequate for assigning one of the other descriptors
- Not likely to be carcinogenic to humans — appropriate when the available data are robust for determining that there is no basis for concern regarding human carcinogenicity.

When a compound's effects vary by exposure route or dose, more than one descriptor can be applied. The narrative descriptions reflect significant advances in cancer risk assessment, but the newer evaluation has not yet been performed for many compounds. Therefore, the 1986 grouping classification is still included in IRIS and is provided here for COPCs that are classified under the previous system. Therefore, consistent with classification information for each chemical on IRIS, both classification systems are provided in Table 5-2.

Table 5-2 provides the cancer-based toxicity information for COPCs for the oral and dermal exposure routes. For the applicable COPCs, Table 5-2 lists the CAS number, oral CSF, TEF for dioxin-like compounds, relative potency factor (RPF) for carcinogenic PAHs, oral absorption efficiency for the dermal pathway, absorbed CSF for the dermal route, mutagenicity classification, weight-of-evidence/cancer guideline description, weight-of-evidence classification system, source, date, toxicity factor tier, surrogate, CAS for surrogate, and rationale/reference for surrogate. Adjustments for dermal absorption are discussed in Section 5.4.

Cancer risks from chemicals that act via a mutagenic MOA are assessed in a manner different from chemicals that do not have a mutagenic MOA (USEPA 2005c). Dose-response values are typically based on the linearized multistage (LMS) model for carcinogens classified as mutagenic; this infers that cancer risks are linear in the low-dose area of the curve (USEPA 2005b, 2005c). Per USEPA's Cancer Guidelines and Supplemental Guidance for Assessing Susceptibility for Early-Life Exposure to Carcinogens (USEPA 2005c), age-dependent adjustment factors (ADAFs) have been applied to risk calculations in this BHHRA for COPCs with a mutagenic MOA. These chemicals include carcinogenic PAHs, chromium(VI), and trichloroethene. Potential contributions to lifetime cancer risk from early-life exposures to these mutagenic COPCs is detailed in the risk characterization (Section 6) and uncertainty evaluation (Section 7). Mutagenic COPCs were identified from the USEPA RSLs (USEPA 2019a). However, per USEPA IRIS (2019b), chromium(VI) has a potentially mutagenic MOA for only the inhalation exposure route; not oral or dermal. Because IRIS does not provide an oral CSF for chromium(VI), a Tier 3 value from NJDEP (2009) is used in the risk assessment. Chromium(VI) is conservatively assessed as a mutagen for the oral and dermal exposure routes in the BHHRA, even though NJDEP's (2009) documentation states that there is no firm evidence of a mutagenic MOA. However, USEPA's draft toxicological review of chromium(VI) (USEPA 2010b) indicates that a mutagenic MOA is relevant to humans and is adequately confirmed in laboratory animals; therefore, early-life susceptibility is relevant, and ADAFs should be applied to the cancer risk calculations. Note that USEPA is in the process of re-evaluating hexavalent chromium using the IRIS process.

As indicated by USEPA (2005c), the following ADAFs are applied to carcinogenic PAHs, trichloroethene, and chromium(VI) in the BHHRA:

- Age <2: ADAF = 10
- Age 2<16: ADAF = 3
- Age ≥16: ADAF = 1

As shown in Table 5-3, age-weighted ADAF values were calculated for the child and adolescent age groupings based on the respective exposure duration. For RME, ADAFs were averaged across the entire age of the receptor. For CTE, exposure was assumed to take place during the latter part of the child's age range. Appendix F (RAGS Part D Table 7 Series — Calculation of Chemical Risks and Noncancer Hazards) shows the chemicals identified as mutagens and details the age-specific ADAFs. The footnotes below the tables show how the ADAFs are incorporated into the cancer risk equation and list the ADAFs applicable to each age group (child, adolescent, adult). TCE is classified by USEPA as being carcinogenic to humans, potentially causing kidney and liver tumors, as well as non-Hodgkin lymphoma (NHL). Evidence indicates a mutagenic mode of action for kidney tumors, but not for liver tumors or NHL. Therefore, USEPA's Toxicological Review of TCE - Support of Summary Information on IRIS (USEPA 2011b) recommends that kidney risk be assessed using the mutagenic approach (ADAF applied), but liver and NHL be addressed using standard cancer equations. Consistent with the LPRSA BHHRA (AECOM 2017), the NBSA BHHRA uses default ADAFs for TCE. Specifically, the age-specific ADAFs are applied to the total lifetime oral CSF of 0.046 mg/kg/day$^{-1}$, instead of using the TCE-specific approach that applies the ADAF to the kidney CSF but not the NHL + liver CSF. As shown in Appendix B-2, the default approach is more stringent (yields higher cancer risks) than the TCE-specific approach discussed by USEPA (2011b). The only other chemical for which USEPA has separate guidance is vinyl chloride, which was not identified as a COPC in any medium.

### 5.4 Gastrointestinal Absorption Efficiency

No dermal toxicity factors are available for the COPCs in this risk assessment; therefore, oral dose-response values are used to evaluate the dermal exposure pathway. The algorithm for estimating dermal absorption gives rise to an absorbed dose, necessitating adjustment of the oral toxicity value to account for an absorbed dose instead of an administered dose. This modification accounts for the absorption efficiency in the gastrointestinal tract in the critical toxicity study that forms the basis of the noncarcinogenic or carcinogenic toxicity factor. For example, in the situation where oral absorption in the critical study is virtually 100% (complete), the absorbed dose is equal to the administered dose; therefore, no adjustment is necessary. Oral absorption efficiency values were obtained from *RAGS Part E, USEPA Supplemental Guidance for Dermal Risk Assessment, Exhibit 4-1* (USEPA 2004b). The oral RfD was multiplied by the oral absorption factor to calculate the dermal RfD. Where no adjustment is recommended, the dermal RfD equals the oral RfD. Similarly, the oral CSF was divided by the oral absorption factor to calculate the dermal CSF. Where no adjustment is recommended, the dermal CSF equals the oral CSF. No adjustment is made for the organic COPCs, because their gastrointestinal absorption is relatively high. As can be seen in Tables 5-1 and 5-2, several of the inorganic COPCs are adjusted for gastrointestinal absorption in deriving the dermal toxicity factors. These inorganics are antimony, cadmium, chromium, manganese, mercury, nickel, silver, and vanadium.

### 5.5 Chemical-Specific Discussion

The toxicity assessment for particular chemicals or chemical classes with unique toxicological considerations in the risk assessment is discussed in the subsections below:

- Dioxins and Furans (Section 5.5.1)
- PCBs (Section 5.5.2)
- PAHs (Section 5.5.3)
- Arsenic (Section 5.5.4)
- Lead (Section 5.5.5)
- Mercury (Section 5.5.6).

### 5.5.1 Dioxins and Furans

Dioxins and furans are determined to be COPCs in fish and crab tissue, accessible surface sediment, and surface water. Because these compounds are present in complex mixtures, the toxicity of 2,3,7,8-TCDD, the most heavily studied of the dioxins and furans, is used as the index for the other members of the group. Seven chlorinated dioxin and ten chlorinated furan congeners bind to the aryl hydrocarbon (Ah) receptor and therefore have a toxic mechanism similar to TCDD. The World Health Organization (WHO) has derived TEFs to normalize the potency of each of the 17 congeners to that of TCDD (Van den Berg et al. 2006). In 2010, USEPA recommended these 2005 WHO consensus TEFs for risk assessment purposes (USEPA 2010a). Specifically, USEPA (2010a) recommends these TEFs be applied for all effects mediated through Ah receptor binding by DLCs, including both cancer and noncancer effects. 2,3,7,8-TCDD has a TEF of 1; USEPA's recommended TEFs are listed in the table below.

| Congener | WHO 2005 TEF |
|---|---|
| *Chlorinated dibenzo-p-dioxins* | |
| 2,3,7,8-TCDD | 1 |
| 1,2,3,7,8-PeCDD | 1 |
| 1,2,3,4,7,8-HxCDD | 0.1 |
| 1,2,3,6,7,8-HxCDD | 0.1 |
| 1,2,3,7,8,9-HxCDD | 0.1 |
| 1,2,3,4,6,7,8-HpCDD | 0.01 |
| OCDD | 0.0003 |
| *Chlorinated dibenzofurans* | |
| 2,3,7,8-TCDF | 0.1 |
| 1,2,3,7,8-PeCDF | 0.03 |
| 2,3,4,7,8-PeCDF | 0.3 |
| 1,2,3,4,7,8-HxCDF | 0.1 |
| 1,2,3,6,7,8-HxCDF | 0.1 |
| 1,2,3,7,8,9-HxCDF | 0.1 |
| 2,3,4,6,7,8-HxCDF | 0.1 |
| 1,2,3,4,6,7,8-HpCDF | 0.01 |

| Congener | WHO 2005 TEF |
|---|---|
| 1,2,3,4,7,8,9-HpCDF | 0.01 |
| OCDF | 0.0003 |
| Source: Van den Berg et al. 2006, USEPA 2010a | |

In this BHHRA, the above TEFs were applied to the 2,3,7,8-TCDD toxicity factor to derive congener-specific CSFs and RfDs (see Tables 5-1 and 5-2). Specifically, the 2,3,7,8-TCDD CSF was multiplied by the TEF to determine the congener-specific CSFs, and the TCDD RfD was divided by the TEF to calculate the congener-specific RfD. Therefore, the EPCs reflect the measured concentration of each congener (no TEF applied), because the TEF was applied later at the toxicity factor step. TCDD-TEQ risks and hazards were calculated by summing the congener-specific risks and hazards. Risks and hazards are presented for individual dioxin/furan congeners, total PCDD/Fs, and total DLCs, which include dioxin-like PCBs (see below).

Potential carcinogenic effects of TCDD-TEQ are evaluated using the USEPA (1997a) HEAST CSF of 150,000 $(mg/kg\text{-}day)^{-1}$, per USEPA (1996b, 2018a, 2018b). The uncertainty evaluation (Section 7.3.6) discusses additional cancer toxicity values that are available for TCDD. Potential noncancer effects of TCDD-TEQ are evaluated using the USEPA IRIS (2019b) RfD of 7E-10 mg/kg-day.

USEPA (2013b) states that the TEF method is most applicable to oral exposures for sediment, soil, fish, and water contaminated with 2,3,7,8-TCDD and DLCs. As an estimate, TEFs may be used for other exposure routes (i.e., inhalation, dermal), but the relative contribution of the various exposure routes to the TEQ should be determined (USEPA 2013b, 2010a). Van den Berg et al. (2006) notes that there is more uncertainty in applying TEFs to abiotic samples (e.g., sediment, surface water) than to biotic media (e.g., fish, crab). USEPA (2013b) recommends that the portion of the total TEQ from 2,3,7,8-TCDD (which has relatively low uncertainty) and from DLCs (which has higher uncertainty) be determined. Under the TEF approach, the TEF of a DLC is assumed to be the same for all end points of concern, for all exposure scenarios, and all are assumed to be full agonists. There is considerable uncertainty associated with these assumptions (USEPA 2010a).

### 5.5.2 Polychlorinated Biphenyls (PCBs)

Total non-dioxin-like PCBs (Total Non-DL PCBs), as well as DL-PCB congeners, are COPCs in fish and crab issue, accessible surface sediment, and surface water. Data on PCB congeners, as well as Aroclors, have been collected during the NBSA RI program. The Aroclor data were not used in the BHHRA but are included in Appendix A as additional information. The total non-DL PCB EPCs were calculated by combining the appropriate concentrations of the individual non-DL PCB congeners. The methods used to estimate carcinogenic risks and noncarcinogenic hazards associated with non-DL PCBs and DL-PCBs in the BHHRA are described in the sections below.

*5.5.2.1 Total Non-Dioxin Like PCBs Approach*

Total non-DL PCBs are evaluated using the USEPA IRIS (2019b) toxicity values for PCB mixtures and certain Aroclors. The method for evaluating carcinogenic effects is detailed first below, followed by noncarcinogenic effects.

Carcinogenic Effects

In IRIS, USEPA (2019b) identified three tiers of oral CSFs for assessment of total PCBs: (1) high risk and persistence, (2) low risk and persistence, and (3) lowest risk and persistence. The selection of a particular CSF varies with PCB chlorine content, as well as exposure route and medium. USEPA recommends the upper-bound oral CSF [2 $(mg/kg\text{-}day)^{-1}$] and central-estimate oral CSF [1 $(mg/kg\text{-}day)^{-1}$] for food-chain exposures (i.e., fish and crab ingestion), as well as sediment ingestion. The PCB CSFs are determined from animal cancer bioassays using PCB mixtures. Therefore, the observed toxic effects are due to combined effects of the mixtures on the whole animal (including dioxin-like toxicity, see below). Based on the range of CSFs in IRIS (USEPA 2019b) and comments from USEPA (2018a), cancer risks from total NDL-PCBs are evaluated as follows:

- All RME Scenarios, Exposure Pathways
  - High risk and persistence, upper-bound CSF of 2 $(mg/kg\text{-}day)^{-1}$ — ingestion of fish and crab, incidental ingestion of sediment, dermal contact with sediment, incidental ingestion of surface water, dermal contact with surface water
- All CTE Scenarios, Exposure Pathways
  - High-risk and persistence, central estimate CSF of 1 $(mg/kg\text{-}day)^{-1}$ — ingestion of fish and crab, incidental ingestion of sediment, dermal contact with sediment, incidental ingestion of surface water, dermal contact with surface water.

Noncarcinogenic Effects

While USEPA has not derived an oral RfD for PCBs as a group, the Agency has performed threshold effect evaluations for the following individual PCB mixtures: Aroclor 1254, 1016, and 1248 (USEPA 2019b). USEPA IRIS (2019b) has published an oral RfD of 2E-05 mg/kg-day (for Aroclor 1254) and an oral RfD of 7E-05 mg/kg-day (for Aroclor 1016). USEPA (2019b) also reviewed Aroclor 1248 data but did not derive an RfD.

The IRIS (USEPA 2019b) Aroclor 1254 RfD is used to estimate the noncancer effects of total NDL-PCBs. While no guidance is available instructing assessors on the choice between the Aroclor 1254 RfD vs. Aroclor 1016 RfD, the oral RfD for the Aroclor that most closely resembles the congener in the environmental media of concern should be used. The RfD for Aroclor 1254, typically used in HHRAs and the more conservative of the two Aroclor toxicity factors, has been chosen for the NBSA BHHRA to assess total NDL-PCBs in all media. USEPA is currently evaluating potential noncarcinogenic effects of PCB mixtures using the IRIS process (USEPA 2019b).

*5.5.2.2 Dioxin-Like PCBs Approach*

Potential risks/hazards have been determined separately for the dioxin-like congeners vs. total non-DL PCBs. The method for evaluating carcinogenic effects is detailed first below, followed by noncarcinogenic effects.

Carcinogenic Effects

A subset of PCB congeners has a mechanism of action similar to that of TCDD (USEPA 1996b, 2010a; Van den Berg et al. 2006). The classification as a "dioxin-like compound" (DLC) is based on a substance's ability to bind to the Ah receptor, as well as similarities in bioaccumulation ability and biochemical characteristics. Twelve coplanar PCBs are identified as being dioxin-like; they each have at least four chlorine atoms with one or zero substitutions at ortho positions. The coplanar PCBs do not have ortho chlorines on either ring, which allows the rings to be positioned in the same plane, but with a flexible conformation. WHO (Van den Berg et al. 2006) derived TEFs to normalize the potency of each of the 12 congeners to that of TCDD, and USEPA (2010a) also recommends these values. USEPA's recommended TEFs are listed in the table below:

| Congener | WHO 2005 TEF |
|---|---|
| *Non-ortho–substituted PCBs* | |
| PCB-77 | 0.0001 |
| PCB-81 | 0.0003 |
| PCB-126 | 0.1 |
| PCB-169 | 0.03 |
| *Mono-ortho–substituted PCBs* | |
| PCB-105 | 0.00003 |
| PCB-114 | 0.00003 |
| PCB-118 | 0.00003 |
| PCB-123 | 0.00003 |
| PCB-156 | 0.00003 |
| PCB-157 | 0.00003 |
| PCB-167 | 0.00003 |
| PCB-189 | 0.00003 |
| Source: Van den Berg et al. 2006, USEPA 2010a | |

Consistent with the TEQ method discussed in Section 5.5.1 for TCDD-TEQ, PCB-TEQs have been calculated using the above TEFs for the 12 coplanar PCBs (USEPA 2010a). Potential cancer risks posed by the DL-PCB congeners are determined using the HEAST (USEPA 1997a) 2,3,7,8-TCDD CSF [150,000

(mg/kg-day)$^{-1}$] and applying the respective TEF. The remaining PCB congeners reported in the analytical data that are not dioxin-like are combined and included in the total non-DL PCB EPCs.

Noncarcinogenic Effects

Similar to the cancer approach, potential noncarcinogenic hazards posed by the DL-PCB congeners are calculated by using the USEPA IRIS (2019b) 2,3,7,8-TCDD RfD of 7E-10 mg/kg-day and applying the congener-specific TEF. As stated in the section above, the remaining PCB congeners reported in the analytical data that are not dioxin-like were combined and included in the total non-DL PCB EPCs.

### 5.5.3 Polycyclic Aromatic Hydrocarbons (PAHs)

Various PAHs are identified as COPCs in biota/tissue, sediment, and surface water. One PAH is not carcinogenic (naphthalene); the other PAHs are the seven carcinogenic PAHs identified by USEPA (1993). The following PAHs have been identified as COPCs in the BHHRA:

| **PAHs as COPCs** | | |
|---|---|---|
| **PAH** | **Carcinogenic/Noncarcinogenic** | **COPC in Medium** |
| Benz(a)anthracene | Carcinogenic | Biota, Sediment, Surface Water |
| Benzo(a)pyrene | Carcinogenic | Biota, Sediment, Surface Water |
| Benzo(b)fluoranthene | Carcinogenic | Biota, Sediment, Surface Water |
| Benzo(k)fluoranthene | Carcinogenic | Sediment, Surface Water |
| Chrysene | Carcinogenic | Biota, Sediment, Surface Water |
| Dibenz(a,h)anthracene | Carcinogenic | Biota, Sediment, Surface Water |
| Indeno(1,2,3-c,d)-pyrene | Carcinogenic | Biota, Sediment, Surface Water |
| Naphthalene | Noncarcinogenic | Surface Water |

Benzo(a)pyrene (BaP) is the most studied of the PAHs; it is the only one of the carcinogenic PAHs with adequate data for USEPA's derivation of toxicity values. USEPA recently re-evaluated BaP toxicity and published a new IRIS file in 2017 (USEPA 2017g, 2019b). The current BaP CSF [(1 mg/kg-d$^{-1}$)] is based on benchmark dose modeling of rodent tumor data. The BaP IRIS file contains an oral RfD (3E-04 mg/kg-d), also based on benchmark dose modeling of rodent data (USEPA 2017g, 2019b). Per USEPA (1993, 2017g), relative potency factors (RPFs) are used to assess the carcinogenic potency of the other six carcinogenic PAHs compared to BaP. The latest USEPA evaluation indicates that BaP is carcinogenic to humans and has a mutagenic MOA (USEPA 2017g). By extension, the other six carcinogenic PAHs are also considered carcinogens (but not human carcinogens) and mutagenic. An oral CSF specific to each carcinogenic PAH is calculated by multiplying the BaP CSF by its RPF. The current USEPA RPFs and calculated CSFs are shown below.

| PAH | Relative Potency Factor (USEPA 1993) | Oral Cancer Slope Factor (mg/kg-day)-1 | CSF Source |
|---|---|---|---|
| Benz(a)anthracene | 0.1 | 1.0E-01 | BaP CSF x RPF |
| Benzo(a)pyrene | 1.0 | 1.0E+00 | USEPA 2019b |
| Benzo(b)fluoranthene | 0.10 | 1.0E-01 | BaP CSF x RPF |
| Benzo(k)fluoranthene | 0.01 | 1.0E-02 | BaP CSF x RPF |
| Chrysene | 0.001 | 1.0E-03 | BaP CSF x RPF |
| Dibenz(a,h)anthracene | 1.0 | 1.0E+00 | BaP CSF x RPF |
| Indeno(1,2,3-c,d)-pyrene | 0.1 | 1.0E-01 | BaP CSF x RPF |
| Naphthalene | -- | -- | -- |

CSF — cancer slope factor
RPF — relative potency factor

As stated above, IRIS provides a BaP oral RfD (USEPA 2019b). Oral RfDs have not been derived for the other carcinogenic PAHs; RPFs are applied to the CSF (USEPA 2017g) and not the RfD.

USEPA IRIS (2019b) provides an oral RfD of 2E-02 mg/kg-day for naphthalene; this value is used in the BHHRA.

### 5.5.4 Arsenic

Crab and fish tissue samples collected from the Newark Bay Study Area were analyzed for total arsenic, which includes both inorganic and organic arsenic, whereas the USEPA cancer slope factor and reference dose values are specifically for the more toxic inorganic arsenic. This distinction is important for assessing the potential risk associated with ingestion of arsenic in crab and fish tissues, because the less toxic organic arsenic predominates in these tissues. The various organic arsenic compounds in fish and crab (e.g., monomethylarsonic acid [MMA)] dimethylarsinic acid [DMA], arsenosugars, arsenobetaine, arsenolipids, arsenocholine) are far less toxic or basically non-toxic to humans (ATSDR 2007).

As discussed in Appendix J, USEPA states in its Technical Summary of Information Available on the Bioaccumulation of Arsenic in Aquatic Organisms that the consensus in the literature is that approximately 10% of arsenic is present as inorganic arsenic in marine fish and shellfish (USEPA 2003c). USEPA (2003c) further states that, while less is known about arsenic speciation in freshwater fish and shellfish, it is believed that 10% or less of arsenic is present as inorganic arsenic. A GSH analysis of LPRSA BHHRA (AECOM 2017) data in blue crab suggests that the average inorganic arsenic proportion is on the order of 1% in those samples. Rough modeling of NBSA arsenic data using LPRSA arsenic data yielded a worst-case tissue value of 2% of total arsenic present as inorganic arsenic (in hepatopancreas). Accordingly, an inorganic proportion of 10% was used in this BHHRA. Therefore, for fish and crab tissues, it was assumed that 10% of total arsenic is in the inorganic form, and 90% is organic arsenic.

Note that arsenic present in sediment and surface water is evaluated as inorganic arsenic. Inorganic arsenic is identified as a COPC in sediment and surface water; both inorganic and organic arsenic are identified as COPCs in biota. For inorganic arsenic, the IRIS oral CSF (1.5E+00 [mg/kg-day]-1) and IRIS oral RfD (3E-04 mg/kg-day) were used (USEPA 2019b). Organic arsenic was evaluated using the ATSDR MRL for DMA (2E-02 mg/kg-day) (ATSDR 2007).

### 5.5.5 Lead

Lead is a COPC in accessible surface sediment, and fish and crab tissues. Appendix E contains the lead evaluation and details the blood lead models used (USEPA 1994a, 1994b, 2003b, 2017d).

### 5.5.6 Mercury

Mercury has been speciated in samples collected from all media—sediment, surface water, and biota—with measurements for mercury and methyl mercury. Mercury is a COPC in sediment, surface water, fish, and crab; methyl mercury is a COPC in fish and crab. Three forms of mercury are applicable to HHRAs:

- Divalent inorganic mercury (usually assumed to be mercuric chloride)
- Methyl mercury (organic mercury)
- Elemental mercury vapor.

The different forms of mercury vary in their health effects and their respective toxicity values.

In sediment, mercury can exist as organic complexes, mercury hydroxide ($Hg(OH)_2$), mercuric chloride ($HgCl_2$), mercuric oxide (HgO), or mercuric sulfide (HgS) (USEPA 1997b). Of these compounds, only $HgCl_2$ has a USEPA-derived toxicity factor. In a water column, the majority of mercury is present as divalent mercury in a complex with dissolved organic carbon; <10% of the total mercury is present as a methyl mercury complex. However, nearly all of the mercury present in fish muscle tissue is in the methylated form (USEPA 1997b). Elemental mercury exposure occurs primarily via inhalation, because it exists as a vapor (USEPA 1997b), but inhalation is not a complete exposure pathway in this BHHRA.

Both mercury and methyl mercury were detected and identified as COPCs in all crab tissue types and all species of fish. Mercury is identified as a COPC in sediment and surface water. Methyl mercury hazards were evaluated using the USEPA IRIS RfD of 1E-04 mg/kg-day (USEPA 2019b); mercury hazards were assessed using the IRIS RfD for mercuric chloride (3E-4 mg/kg-day) (USEPA 2019b).

# 6. Risk Characterization

In the risk characterization step of the BHHRA, possible threats to human health related to potential exposure to COPCs in environmental media are determined. Specifically, the quantitative exposure factors derived in the Exposure Assessment (Section 4.0) are combined with the chemical-specific toxicity factors for COPCs (Section 5.0). In assessing carcinogenic effects, estimated intakes and cancer toxicity factors are integrated to calculate the probability that a person will develop cancer; in determining noncarcinogenic effects, projected intakes of COPCs are compared to the noncarcinogenic toxicity factor. Cancer risk is determined by averaging exposure over a 70-year lifetime; noncarcinogenic hazard is assessed by averaging exposure over the exposure duration (USEPA 1989).

Potential cancer risks and noncancer hazards are determined using different methods. This variation is due to the assumption that potential carcinogens act by a no-threshold MOA, but noncarcinogens are assumed to have a threshold; a level below which no response is expected to occur. Thus, in the cancer assessment, risk of developing cancer is calculated using the CSF, while in the noncarcinogenic evaluation, it can be determined whether the dose is above or below the threshold concentration (e.g., RfD). CSFs and RfDs are outlined in Section 5.0. The method for characterizing carcinogenic risks is discussed in Section 6.1, and the steps to characterize noncarcinogenic hazards are found in Section 6.2. Section 6.3 displays the results of the risk characterization. Section 6.4 discusses selection of potential Chemicals of Concern (potential COCs). Risk and hazard calculations are presented in RAGS Part D format (Table 7 series) in Appendix F.

## 6.1 Carcinogenic Risk Characterization

The carcinogenic risk characterization predicts the upper-bound probability that a person will develop cancer during their lifetime due to exposure to a COPC in a particular environmental medium or combined environmental media. This probability is a function of the dose of the COPC (see Exposure Assessment, Section 5.0) and the chemical-specific toxicity value (CSF) (see Toxicity Assessment, Section 4.0). The excess lifetime cancer risk (ELCR) is the incremental chance of developing cancer as a result of exposure to site-related COPCs.

The equation below shows how the ELCR is calculated from the estimated lifetime average daily dose (LADD) of a COPC (USEPA1989):

$$ELCR = 1 - e^{(-CSF\ x\ Lifetime\ intake)} \qquad \text{Equation 6-1}$$

where:

ELCR = excess lifetime cancer risk

CSF = cancer slope factor

When CSF multiplied by the lifetime intake greatly exceeds 1, the ELCR approaches 1 (100% probability of developing cancer). Alternatively, when CSF multiplied by the lifetime intake is <0.01 (1/100 chance of

cancer occurring), Equation 6-1 is approximated by Equation 6-2, which was used for calculating cancer risks in this BHHRA:

$$ELCR = Lifetime\ intake\ (mg/kg-day)\ x\ CSF\ (mg/kg-day)^{-1} \qquad \text{Equation 6-2}$$

where:

ELCR = excess lifetime cancer risk

CSF = cancer slope factor

The ELCR is a unitless, upper-bound estimate of the cancer risk potentially resulting from a person's exposure to that COPC by a particular exposure pathway. For mutagenic COPCs, the LADD is multiplied by the age-appropriate ADAF (see Section 5.3).

For a receptor, total cancer risks are calculated by adding risks across all applicable chemicals and exposure pathways. In addition to calculation of total risks for each receptor age group, the child and adult age group cancer risks are added together. This cancer risk across age groups represents potential total risks to a person who is exposed to site COPC(s) over the entire residential exposure duration of 26 years (6 years as a child plus 20 years as an adult for the RME scenario; for the CTE scenario, 3 years as a child plus 9 years as an adult, for a total of 12 years). The adult-plus-child scenario was included for cancer risks at the request of USEPA (2017a), in lieu of including an age-adjusted scenario. Carcinogenic risks for a combined adult/child receptor are estimated for the angler, swimmer, and wader scenarios.

Although it is generally accepted that all potential carcinogens do not likely affect the same target organ(s) nor act by the same MOA, human health risk assessments assume that cancer risks are cumulative or additive (USEPA 2005b). For each receptor, risks have been added across chemicals and exposure pathways to calculate the potential total site cancer risk for the particular receptor, regardless of the target organs for the various carcinogenic COPCs.

USEPA guidance (1991d) provides recommendations on target risk levels for reviewing risk assessment cancer-based results. Per USEPA (1991d), "The upper boundary of the risk range is not a discrete line at $10^{-4}$, although USEPA generally considered acceptable if justified based on site-specific conditions." and "Where the cumulative carcinogenic site risk to an individual based on reasonable maximum exposure for both current and future land use is less than $10^{-4}$, and the noncarcinogenic hazard quotient is less than 1, action generally is not warranted unless there are adverse environmental impacts."

The total ELCR for each receptor is evaluated against the NCP risk range of $10^{-6}$ to $10^{-4}$ (i.e., cancer risk of one in one million to one in ten thousand) (USEPA 1990). Based on USEPA (1991d), a cumulative cancer risk level of $10^{-4}$ will be used to evaluate the total risks in the BHHRA. If a receptor's total cancer risk is below $10^{-4}$, no further action or evaluation is believed to be necessary (based on potential cancer risks). However, if a receptor's total cancer risk is greater than the acceptable risk range of $10^{-4}$, COPCs with individual

pathway risks exceeding $10^{-6}$ become potential COCs. Note that cumulative noncarcinogenic hazards must also be examined in determining the possible need for remediation (see below).

### 6.2 Noncarcinogenic Risk Characterization

The HQ is a unitless ratio calculated to determine a chemical's potential to result in noncarcinogenic health effects at a level of concern. The receptor-specific HQ is calculated by dividing the chronic intake by the RfD for a certain COPC, as shown in the following equation:

$$HQ = \frac{Chronic\ Intake\ (mg/kg-day)}{RfD\ (mg/kg-day)}$$

where:

HQ = hazard quotient

RfD = reference dose

An HQ ≤1 indicates that chronic intake does not exceed the RfD and adverse noncancer health effects are not anticipated (USEPA 1989, 1991d). While an HQ that exceeds 1 may indicate the possibility of adverse noncancer effects occurring, the level of exceedance cannot be correlated directly to an effect level or likelihood.

The HQs for the individual COPCs are summed to calculate the total hazard index (HI). Receptor-specific, total site HIs are calculated by adding the HIs for the exposure pathways relevant to the particular receptor. Per USEPA guidance, HIs by target organ are also calculated, because noncancer effects are typically summed by target organ (see RAGS D Table 9 and 10 series [Appendix G and I, respectively]) (USEPA 1989).

Each COPC that causes an exceedance of the HI of 1 for a particular receptor and for a particular target organ effect is designated a potential COC. Where the cumulative target organ effect HIs for a receptor are less than 1, then no further evaluation or action will be recommended based on potential noncarcinogenic hazards (carcinogenic risks must also be considered as discussed above). Where the cumulative target organ effect HI for a receptor is greater than 1, the goal of protection of an HI equal to 1 has been exceeded. In these cases, potential COCs are identified as those COPCs with individual pathway HQs greater than 0.1.

#### 6.2.1 Risk Characterization for Lead

Lead was identified as a COPC in accessible surface sediment and crab muscle/hepatopancreas tissue. Predicted blood lead levels (PbBs) for children and adults are compared to 5 micrograms per deciliter (µg/dL), which is USEPA's target blood lead level used in the ALM spreadsheet (USEPA 2017d). The OLEM Directive 9200.2-167 (USEPA 2016b) highlights EPA's Office of Land and Emergency Management review of the health effects evidence for lead in the 2013 Integrated Science Assessment for Lead (ISA for Lead)

and found that several studies have observed "clear evidence of cognitive function decrements (as measured by Full Scale IQ, academic performance, and executive function) in young children (4 to 11 years old) with mean or group blood Pb levels between 2 and 8 µg/dL (measured at various life stages and time periods). Consistent with the Regional policy implementing this Memo, a blood lead level of 5 ug/dL is used in assessments using the Adult Lead Model and the Integrated Exposure Uptake and Biokinetic Model. The lead risk assessment is presented in Appendix E; the results for each receptor are summarized in Section 6.3.6.

## 6.3 Risk Characterization Results

The results of the risk characterization for each receptor are presented below. Estimated cancer risks exceeding $10^{-4}$ and/or noncancer hazard indices exceeding 1 are highlighted. The supporting calculations for both the RME and CTE scenarios are presented in the RAGS Part D Table 7 format in Appendix F, including COPC-specific risks and hazards for each receptor, medium, and exposure pathway. Additionally, the analysis of HIs greater than 1 on a target-organ basis for both the RME and CTE scenarios are presented in the RAGS Part D Table 9 format in Appendix G. Finally, tables showing the percent contribution of each COPC to the total risk are presented in Appendix H for fish or crab consumption by the angler/sportsman only, because the risks/hazards associated with exposure to sediment and surface water are on the order of 100-fold lower or more than those associated with fish or crab consumption.

It is important to note that two sets of cancer risks and noncancer hazards were estimated: (1) using USEPA's Kaplan-Meier (KM) calculator to derive the TEQ concentrations for PCDDs and DL-PCBs (Version 9.1; issued July 2014) and applying the toxicity criteria for 2,3,7,8-TCDD, and (2) manually calculating the TEQ DF and TEQ PCB based on the concentration of each congener and applying the appropriate TEF to the toxicity criteria for 2,3,7,8-TCDD in the risk calculation. The former cancer risk values are referred to in the text and tables as "Total PCDD/Fs (based on KM TEQ)" and "Total DL-PCBs (based on KM TEQ)," whereas the latter values are referred to as "Total PCDD/Fs (excluding KM TEQ)" and "Total DL-PCBs (excluding KM TEQ)." Cumulative risk and hazard estimates are also designated "Total (based on KM TEQ)" or "Total (excluding KM TEQ)." Both sets of estimates are presented in the RAGS Part D tables (Appendix F for Table 7s and Appendix G for Table 9s), and in the receptor-specific tables below. The purpose of this dual approach was to allow evaluation of which specific congeners were the predominant risk drivers, which would not be possible by estimating risks/hazards based on the KM TEQ alone. As discussed in the following sections, there is essentially no difference in the risk/hazard estimates between the two methods.

### 6.3.1 Angler/Sportsman

Anglers/sportsmen are assumed to be exposed to COPCs in fish or crab self-caught in the NBSA via ingestion, to COPCs in accessible surface sediment and surface water via dermal contact and incidental ingestion. Three age groups were evaluated: a child (1 to <7 years), an adolescent (7 to 19 years), and an adult (>18 years). While children were assumed to consume fish or crab caught by an adult or adolescent angler/sportsman, exposure to sediment and surface water was not evaluated for a child angler/sportsman, because (1) children would not be expected to accompany adolescents or adults very often, if at all, due to

safety concerns, and (2) any exposure would be less than that experienced by children who visit the NBSA to wade or swim.

Potential cancer risks and noncancer hazards associated with adolescent and adult angler/sportsman exposure to accessible surface sediment and surface water were estimated on a site-wide (or Bay-wide) basis. These values are summed with those for ingestion of fish or crab to estimate cumulative cancer risks and noncancer hazards for these receptors. Cumulative site-wide cancer risks and noncancer hazards for the angler/sportsman receptor are presented in Tables 6-3 through 6-6; results for each age group are discussed below.

*6.3.1.1 Angler/Sportsman — Child*

The following table summarizes the cumulative potential cancer risks and noncancer HIs for the child angler/sportsman. Values that exceed the NCP risk range of $10^{-6}$ to $10^{-4}$ or an HI of 1 (USEPA 1991d) are bolded.

| **Angler/Sportsman Child (Age 1 to <7)** | | | | | | |
|---|---|---|---|---|---|---|
| Media | RAGS D Table | Scenario | RME | | CTE | |
| | | | Total Risk | Total HI | Total Risk | Total HI |
| Fish Tissue | 9.3 | Mixed Fish Diet (a) | | | | |
| | | Total (excluding KM TEQs) (b) | **3E-04** | **4E+01** | 9E-06 | **4E+00** |
| | | Total (based on KM TEQs) (c) | **3E-04** | **4E+01** | 9E-06 | **4E+00** |
| Crab Tissue | 9.3 | Crab Muscle & Hepatopancreas | | | | |
| | | Total without KM TEQ (b) | **3E-04** | **5E+01** | 2E-05 | **7E+00** |
| | | Total with KM TEQ (c) | **3E-04** | **5E+01** | 2E-05 | **7E+00** |

(a) Mixed fish diet composed of equal fractions of American eel, bluefish, striped bass, summer flounder, and white perch.
(b) Cumulative cancer risks and hazard indices where TEQ is calculated manually.
(c) Cumulative cancer risks and hazard indices where TEQ is calculated using the KM TEQ calculator.

For the RME scenario, the cumulative total potential cancer risk for the child angler/sportsman who consumes a mixed fish diet or a crab muscle and hepatopancreas diet is approximately $3\times10^{-4}$ for both TEQ approaches, which exceeds the NCP risk range. The primary contributors to these exceedances are 2,3,7,8-TCDD, PCB-126, and non-DL PCBs, in fish and crab tissue (see Appendix H). The cumulative total potential cancer risks for the CTE scenario are within the NCP risk range.

The cumulative total potential noncancer HIs for the RME scenario are 40 for fish consumption and 50 for crab consumption, for both TEQ approaches, which exceed the noncancer goal of an HI of 1 (USEPA 1991d). The primary contributors to these exceedances are also 2,3,7,8-TCDD, PCB-126, and non-DL PCBs in fish tissue and 2,3,7,8-TCDD, copper, PCB-126, and non-DL PCBs in crab tissue. For the CTE scenario, the total potential noncancer HI associated with fish consumption is 4 and for crab consumption is 7 for both TEQ approaches. These values also exceed the noncancer goal. The same chemicals—2,3,7,8-

TCDD, copper (crab tissue only), PCB-126, and non-DL PCBs—are the primary contributors to these exceedances (see Appendix H).

*6.3.1.2 Angler/Sportsman — Adolescent*

The following table summarizes the cumulative potential cancer risks and noncancer HIs for the adolescent angler/sportsman. Values that exceed the NCP risk range of $10^{-6}$ to $10^{-4}$ or an HI of 1 (USEPA 1991d) are bolded.

| **Angler/Sportsman Adolescent (Age 7 to <19)** | | | | | | |
|---|---|---|---|---|---|---|
| Media | RAGS D Table | Scenario | RME | | CTE | |
| | | | Total Risk | Total HI | Total Risk | Total HI |
| Accessible Surface Sediment | 9.2 | Baywide | | | | |
| | | Total (excluding KM TEQs) (b) | 2E-06 | 1E-01 | 4E-07 | 4E-02 |
| | | Total (based on KM TEQs) (c) | 2E-06 | 1E-01 | 4E-07 | 4E-02 |
| Surface Water | 9.2 | Baywide | | | | |
| | | Total (excluding KM TEQs) (b) | 8E-08 | 2E-03 | 8E-09 | 5E-04 |
| | | Total (based on KM TEQs) (c) | 8E-08 | 2E-03 | 8E-09 | 5E-04 |
| Fish Tissue | 9.2 | Mixed Fish Diet (a) | | | | |
| | | Total (excluding KM TEQs) (b) | **3E-04** | **3E+01** | 1E-05 | **2E+00** |
| | | Total (based on KM TEQs) (c) | **3E-04** | **3E+01** | 1E-05 | **2E+00** |
| Crab Tissue | 9.2 | Crab Muscle & Hepatopancreas | | | | |
| | | Total (excluding KM TEQs) (b) | **4E-04** | **3E+01** | 2E-05 | **4E+00** |
| | | Total (based on KM TEQs) (c) | **3E-04** | **3E+01** | 2E-05 | **4E+00** |
| Baywide Cumulative Total Risk/Hazard | 9.2 | Mixed Fish Diet (a) | | | | |
| | | Total (excluding KM TEQs) (b) | **3E-04** | **3E+01** | 1E-05 | **2E+00** |
| | | Total (based on KM TEQs) (c) | **3E-04** | **3E+01** | 1E-05 | **2E+00** |
| | 9.2 | Crab Muscle & Hepatopancreas | | | | |
| | | Total (excluding KM TEQs) (b) | **4E-04** | **3E+01** | 2E-05 | **4E+00** |
| | | Total (based on KM TEQs) (c) | **3E-04** | **3E+01** | 2E-05 | **4E+00** |

(a) Mixed fish diet composed of equal fractions of American eel, bluefish, striped bass, summer flounder, and white perch.
(b) Cumulative cancer risks and hazard indices where TEQ is calculated manually.
(c) Cumulative cancer risks and hazard indices where TEQ is calculated using the KM TEQ calculator.

For the RME scenario, the cumulative total potential cancer risk for the adolescent angler/sportsman who consumes a mixed fish diet is approximately $3\times10^{-4}$ for both TEQ approaches, and for a crab muscle and hepatopancreas diet, is approximately $4\times10^{-4}$ when the TEQ is calculated manually and $3\times10^{-4}$ when the TEQ is calculated using the KM TEQ calculator. All of these values exceed the NCP risk range. The primary contributors to these exceedances are 2,3,7,8-TCDD, PCB-126, and non-DL PCBs in fish and crab tissue (see Appendix H). Direct-contact exposure to accessible surface sediment and surface water contributes only a small amount to the cumulative cancer risk, with risk estimates within or below the NCP risk range. The cumulative total potential cancer risks for the CTE scenario are within the NCP risk range.

The cumulative total potential noncancer HI for the RME scenario is 30 for fish consumption and 30 for crab consumption, for both TEQ approaches, which exceed the noncancer goal of an HI of 1 (USEPA 1991d). The primary contributors to these exceedances are also 2,3,7,8-TCDD, PCB-126, and non-DL PCBs in fish tissue and 2,3,7,8-TCDD, copper, PCB-126, and non-DL PCBs in crab tissue. For the CTE scenario, the total potential noncancer HI associated with fish consumption is 2, and for crab consumption is 4, for both TEQ approaches. These values also exceed the noncancer goal. The same chemicals—2,3,7,8-TCDD, copper (crab tissue only), PCB-126, and non-DL PCBs—are the primary contributors to these exceedances (see Appendix H).

#### *6.3.1.3 Angler/Sportsman — Adult*

The following table summarizes the cumulative potential cancer risks and noncancer HIs for the adult angler/sportsman. Values that exceed the NCP risk range of $10^{-6}$ to $10^{-4}$ or an HI of 1 (USEPA 1991d) are bolded.

| Angler/Sportsman Adult | | | | | | |
|---|---|---|---|---|---|---|
| Media | RAGS D Table | Scenario | RME | | CTE | |
| | | | Total Risk | Total HI | Total Risk | Total HI |
| Accessible Surface Sediment | 9.1 | Baywide | | | | |
| | | Total (excluding KM TEQs) (b) | 4E-06 | 1E-01 | 7E-07 | 4E-02 |
| | | Total (based on KM TEQs) (c) | 4E-06 | 1E-01 | 6E-07 | 4E-02 |
| Surface Water | 9.1 | Baywide | | | | |
| | | Total (excluding KM TEQs) (b) | 5E-08 | 1E-03 | 5E-09 | 4E-04 |
| | | Total (based on KM TEQs) (c) | 5E-08 | 1E-03 | 5E-09 | 4E-04 |
| Fish Tissue | 9.1 | Mixed Fish Diet (a) | | | | |
| | | Total (excluding KM TEQs) (b) | **5E-04** | **3E+01** | 2E-05 | **2E+00** |
| | | Total (based on KM TEQs) (c) | **6E-04** | **3E+01** | 2E-05 | **2E+00** |
| Crab Tissue | 9.1 | Crab Muscle & Hepatopancreas | | | | |
| | | Total (excluding KM TEQs) (b) | **6E-04** | **3E+01** | 4E-05 | **4E+00** |
| | | Total (based on KM TEQs) (c) | **6E-04** | **3E+01** | 3E-05 | **4E+00** |
| Baywide Cumulative Total Risk/Hazard | 9.1 | Mixed Fish Diet (a) | | | | |
| | | Total (excluding KM TEQs) (b) | **5E-04** | **3E+01** | 2E-05 | **2E+00** |
| | | Total (based on KM TEQs) (c) | **6E-04** | **3E+01** | 2E-05 | **2E+00** |
| | 9.1 | Crab Muscle & Hepatopancreas | | | | |
| | | Total (excluding KM TEQs) (b) | **6E-04** | **3E+01** | 4E-05 | **4E+00** |
| | | Total (based on KM TEQs) (c) | **6E-04** | **3E+01** | 4E-05 | **4E+00** |

(a) Mixed fish diet composed of equal fractions of American eel, bluefish, striped bass, summer flounder, and white perch.
(b) Cumulative cancer risks and hazard indices where TEQ is calculated manually.
(c) Cumulative cancer risks and hazard indices where TEQ is calculated using the KM TEQ calculator.

For the RME scenario, the cumulative total potential cancer risk for the adult angler/sportsman who consumes a mixed fish diet is approximately $5\times10^{-4}$ when the TEQ is calculated manually and $6\times10^{-4}$ when the TEQ is calculated using the KM TEQ calculator, and for a crab muscle and hepatopancreas diet, is approximately $6\times10^{-4}$ for both TEQ approaches. All of these values exceed the NCP risk range. The primary contributors to these exceedances are 2,3,7,8-TCDD, PCB-126, and non-DL PCBs, in fish and crab tissue (see Appendix H). Direct-contact exposure to accessible surface sediment and surface water contributes only a small amount to the cumulative cancer risk, with risk estimates within or below the NCP risk range. The cumulative total potential cancer risks for the CTE scenario are within the NCP risk range.

The cumulative total potential noncancer HI for the RME scenario is 30 for fish consumption and 30 for crab consumption, for both TEQ approaches, which exceed the noncancer goal of an HI of 1 (USEPA 1991d). The primary contributors to these exceedances are also 2,3,7,8-TCDD, PCB-126, and non-DL PCBs in fish tissue and 2,3,7,8-TCDD, copper, PCB-126, and non-DL PCBs in crab tissue. For the CTE scenario, the total potential noncancer HI associated with fish consumption is 2, and for crab consumption is 4, for both TEQ approaches. These values also exceed the noncancer goal. The same chemicals—2,3,7,8-TCDD, copper (crab tissue only) PCB-126, and non-DL PCBs—are the primary contributors to these exceedances (see Appendix H).

*6.3.1.4 Angler/Sportsman — Combined Adult/Child*

As discussed in Section 6.1, a combined adult and child receptor is evaluated for purposes of estimating total cancer risks assuming that exposure occurs over the entire exposure duration for a resident. This equates to 6 years as a child and 20 years as an adult, for a total of 26 years, for the RME scenario, and 3 years as a child and 9 years as an adult, for a total of 12 years, for the CTE scenario. The following table summarizes the cumulative potential cancer risks for the combined adult/child angler/sportsman. Values that exceed the NCP risk range of $10^{-6}$ to $10^{-4}$ are bolded.

| **Angler/Sportsman Combined Adult/Child (Cancer only) (d)** | | | | |
|---|---|---|---|---|
| Media | RAGS D Table | Scenario | RME Total Risk | CTE Total Risk |
| Accessible Surface Sediment | 9.A | Baywide | | |
| | | Total (excluding KM TEQs) (b) | 4E-06 | 7E-07 |
| | | Total (based on KM TEQs) (c) | 4E-06 | 6E-07 |
| Surface Water | 9.A | Baywide | | |
| | | Total (excluding KM TEQs) (b) | 5E-08 | 5E-09 |
| | | Total (based on KM TEQs) (c) | 5E-08 | 5E-09 |
| Fish Tissue | 9.A | Mixed Fish Diet (a) | | |
| | | Total (excluding KM TEQs) (b) | **8E-04** | 3E-05 |
| | | Total (based on KM TEQs) (c) | **8E-04** | 3E-05 |
| Crab Tissue | 9.A | Crab Muscle & Hepatopancreas | | |
| | | Total (excluding KM TEQs) (b) | **8E-04** | 5E-05 |
| | | Total (based on KM TEQs) (c) | **8E-04** | 5E-05 |
| Baywide Cumulative Total Risk/Hazard | 9.A | Mixed Fish Diet (a) | | |
| | | Total (excluding KM TEQs) (b) | **8E-04** | 3E-05 |
| | | Total (based on KM TEQs) (c) | **8E-04** | 3E-05 |
| | 9.A | Crab Muscle & Hepatopancreas | | |
| | | Total (excluding KM TEQs) (b) | **8E-04** | 5E-05 |
| | | Total (based on KM TEQs) (c) | **8E-04** | 5E-05 |

(a) Mixed fish diet composed of equal fractions of American eel, bluefish, striped bass, summer flounder, and white perch.
(b) Cumulative cancer risks where TEQ calculated manually.
(c) Cumulative cancer risks TEQ calculated using the KM TEQ calculator.
(d) Potential cancer risks in this table represent exposures for a child and adult over a 26-year period for RME and a 12-year exposure duration for CTE.

For the RME scenario, the cumulative total potential cancer risk for the combined adult/child angler/sportsman who consumes a mixed fish diet or a crab muscle and hepatopancreas diet is approximately $8\times10^{-4}$ for both TEQ approaches, which exceeds the NCP risk range. The primary contributors to these exceedances are 2,3,7,8-TCDD, PCB-126, and non-DL PCBs in fish and crab tissue (see Appendix H). Direct-contact exposure to accessible surface sediment and surface water by the adult angler/sportsman contributes only a small amount to the cumulative cancer risk, with risk estimates within or

below the NCP risk range. The cumulative total potential cancer risks for the CTE scenario are within the NCP risk range.

### 6.3.2 Swimmer

Swimmers are assumed to be exposed to COPCs in accessible surface sediment and surface water via dermal contact and incidental ingestion. Three age groups were evaluated: a child (1 to <7 years), an adolescent (7 to 19 years), and an adult (>18 years). In addition, a combined adult/child receptor was evaluated for carcinogenic effects only, assuming a total exposure duration of 26 years for the RME scenario and 12 years for the CTE scenario.

Potential cancer risks and noncancer hazards associated with child, adolescent, and adult swimmer exposure to accessible surface sediment and surface water were estimated on a site-wide (or Bay-wide) basis. These values are summed to estimate cumulative cancer risks and noncancer hazards for these receptors. Cumulative sitewide cancer risks and noncancer hazards for the swimmer receptor are presented in Tables 6-1 and 6-2, respectively; results for each age group are discussed below.

#### *6.3.2.1 Swimmer — Child*

The following table summarizes the cumulative potential cancer risks and noncancer HIs for the child swimmer. The cumulative total potential cancer risks and noncancer hazards for this receptor are within or below the NCP risk range and below an HI of 1 (USEPA 1991d) for both the RME and CTE scenarios, regardless of TEQ approach.

| **Swimmer Child (Age 1 to <7)** | | | | | | |
|---|---|---|---|---|---|---|
| Media | RAGS D Table | Scenario | RME | | CTE | |
| | | | Total Risk | Total HI | Total Risk | Total HI |
| Accessible Surface Sediment | 9.7 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 1E-06 | 2E-01 | 2E-07 | 5E-02 |
| | | Total (based on KM TEQs) (b) | 1E-06 | 2E-01 | 2E-07 | 5E-02 |
| Surface Water | 9.7 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 2E-07 | 8E-03 | 4E-08 | 4E-03 |
| | | Total (based on KM TEQs) (b) | 2E-07 | 8E-03 | 4E-08 | 4E-03 |
| Baywide Cumulative Total Risk/Hazard | 9.7 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 2E-06 | 2E-01 | 2E-07 | 5E-02 |
| | | Total (based on KM TEQs) (b) | 2E-06 | 1E-01 | 2E-07 | 5E-02 |

(a) Cumulative cancer risks and hazard indices where TEQ is calculated manually.
(b) Cumulative cancer risks and hazard indices where TEQ is calculated using the KM TEQ calculator.

*6.3.2.2 Swimmer — Adolescent*

The following table summarizes the cumulative potential cancer risks and noncancer HIs for the adolescent swimmer. The cumulative total potential cancer risks and noncancer hazards for this receptor are within or below the NCP risk range and below an HI of 1 (USEPA 1991d) for both the RME and CTE scenarios, regardless of TEQ approach.

| **Swimmer Adolescent (Age 7 to <19)** | | | | | | |
|---|---|---|---|---|---|---|
| Media | RAGS D Table | Scenario | RME | | CTE | |
| | | | Total Risk | Total HI | Total Risk | Total HI |
| Accessible Surface Sediment | 9.6 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 2E-06 | 1E-01 | 3E-07 | 3E-02 |
| | | Total (based on KM TEQs) (b) | 2E-06 | 1E-01 | 3E-07 | 3E-02 |
| Surface Water | 9.6 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 5E-07 | 1E-02 | 1E-07 | 6E-03 |
| | | Total (based on KM TEQs) (b) | 5E-07 | 1E-02 | 1E-07 | 6E-03 |
| Baywide Cumulative Total Risk/Hazard | 9.6 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 2E-06 | 1E-01 | 4E-07 | 4E-02 |
| | | Total (based on KM TEQs) (b) | 2E-06 | 1E-01 | 4E-07 | 4E-02 |

(a) Cumulative cancer risks and hazard indices where TEQ is calculated manually.
(b) Cumulative cancer risks and hazard indices where TEQ is calculated using the KM TEQ calculator.

*6.3.2.3 Swimmer — Adult*

The following table summarizes the cumulative potential cancer risks and noncancer HIs for the adult swimmer. The cumulative total potential cancer risks and noncancer hazards for this receptor are within or below the NCP risk range and below an HI of 1 (USEPA 1991d) for both the RME and CTE scenarios, regardless of TEQ approach.

| Swimmer<br>Adult | | | | | | |
|---|---|---|---|---|---|---|
| Media | RAGS D Table | Scenario | RME | | CTE | |
| | | | Total Risk | Total HI | Total Risk | Total HI |
| Accessible Surface Sediment | 9.5 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 1E-06 | 3E-02 | 2E-07 | 1E-02 |
| | | Total (based on KM TEQs) (b) | 1E-06 | 3E-02 | 2E-07 | 1E-02 |
| Surface Water | 9.5 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 9E-08 | 3E-03 | 2E-08 | 1E-03 |
| | | Total (based on KM TEQs) (b) | 9E-08 | 3E-03 | 2E-08 | 1E-03 |
| Baywide Cumulative Total Risk/Hazard | 9.5 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 1E-06 | 3E-02 | 2E-07 | 1E-02 |
| | | Total (based on KM TEQs) (b) | 1E-06 | 3E-02 | 2E-07 | 1E-02 |

(a) Cumulative cancer risks and hazard indices where TEQ is calculated manually.
(b) Cumulative cancer risks and hazard indices where TEQ is calculated using the KM TEQ calculator.

*6.3.2.4 Swimmer — Combined Adult/Child*

The following table summarizes the cumulative potential cancer risks for the combined adult/child swimmer. The cumulative total potential cancer risks for this receptor are within or below the NCP risk range for both the RME and CTE scenarios, regardless of TEQ approach.

| Swimmer<br>Combined Adult/Child (Cancer only) (a) | | | | |
|---|---|---|---|---|
| Media | RAGS D Table | Scenario | RME | CTE |
| | | | Total Risk | Total Risk |
| Accessible Surface Sediment | 9.B | Baywide | | |
| | | Total (excluding KM TEQs) (b) | 2E-06 | 3E-07 |
| | | Total (based on KM TEQs) (c) | 2E-06 | 3E-07 |
| Surface Water | 9.B | Baywide | | |
| | | Total (excluding KM TEQs) (b) | 3E-07 | 5E-08 |
| | | Total (based on KM TEQs) (c) | 3E-07 | 4E-08 |
| Baywide Cumulative Total Risk/Hazard | 9.B | Mixed Fish Diet (a) | | |
| | | Total (excluding KM TEQs) (b) | 3E-06 | 4E-07 |
| | | Total (based on KM TEQs) (c) | 3E-06 | 4E-07 |

(a) Potential cancer risks in this table represent exposures for a child and adult over a 26-year period for RME and a 12-year exposure duration for CTE.
(b) Cumulative cancer risks where TEQ is calculated manually.
(c) Cumulative cancer risks where TEQ is calculated using the KM TEQ calculator.

### 6.3.3 Wader

Waders are assumed to be exposed to COPCs in accessible surface sediment and surface water via dermal contact and incidental ingestion. Three age groups were evaluated: a child (1 to <7 years), an adolescent (7 to 19 years), and an adult (>18 years). In addition, a combined adult/child receptor was evaluated for carcinogenic effects only, assuming a total exposure duration of 26 years for the RME scenario and 12 years for the CTE scenario.

Potential cancer risks and noncancer hazards associated with child, adolescent, and adult wader exposure to accessible surface sediment and surface water were estimated on a site-wide (or Bay-wide) basis. These values are summed to estimate cumulative cancer risks and noncancer hazards for these receptors. Cumulative sitewide cancer risks and noncancer hazards for the wader receptor are presented in Tables 6-1 and 6-2, respectively; results for each age group are discussed below.

#### *6.3.3.1 Wader — Child*

The following table summarizes the cumulative potential cancer risks and noncancer HIs for the child wader. The cumulative total potential cancer risks and noncancer hazards for this receptor are within or below the NCP risk range and below an HI of 1 (USEPA 1991d) for both the RME and CTE scenarios, regardless of TEQ approach.

| **Wader<br>Child (Age 1 to <7)** | | | | | | |
|---|---|---|---|---|---|---|
| Media | RAGS D Table | Scenario | RME | | CTE | |
| | | | Total Risk | Total HI | Total Risk | Total HI |
| Accessible Surface Sediment | 9.7 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 1E-06 | 2E-01 | 2E-07 | 5E-02 |
| | | Total (based on KM TEQs) (b) | 1E-06 | 2E-01 | 2E-07 | 5E-02 |
| Surface Water | 9.7 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 3E-08 | 1E-03 | 1E-08 | 4E-04 |
| | | Total (based on KM TEQs) (b) | 3E-08 | 1E-03 | 1E-08 | 4E-04 |
| Baywide Cumulative Total Risk/Hazard | 9.7 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 2E-06 | 2E-01 | 2E-07 | 5E-02 |
| | | Total (based on KM TEQs) (b) | 1E-06 | 2E-01 | 2E-07 | 5E-02 |
| (a) Cumulative cancer risks and hazard indices where TEQ is calculated manually.<br>(b) Cumulative cancer risks and hazard indices where TEQ is calculated using the KM TEQ calculator. | | | | | | |

#### *6.3.3.2 Wader — Adolescent*

The following table summarizes the cumulative potential cancer risks and noncancer HIs for the adolescent wader. The cumulative total potential cancer risks and noncancer hazards for this receptor are within or

below the NCP risk range and below an HI of 1 (USEPA 1991d) for both the RME and CTE scenarios, regardless of TEQ approach.

| Wader<br>Adolescent (Age 7 to <19) | | | | | | |
|---|---|---|---|---|---|---|
| Media | RAGS D Table | Scenario | RME | | CTE | |
| | | | Total Risk | Total HI | Total Risk | Total HI |
| Accessible Surface Sediment | 9.6 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 2E-06 | 1E-01 | 3E-07 | 3E-02 |
| | | Total (based on KM TEQs) (b) | 2E-06 | 1E-01 | 3E-07 | 3E-02 |
| Surface Water | 9.6 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 6E-08 | 2E-03 | 7E-09 | 4E-04 |
| | | Total (based on KM TEQs) (b) | 6E-08 | 2E-03 | 7E-09 | 4E-04 |
| Baywide Cumulative Total Risk/Hazard | 9.6 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 2E-06 | 1E-01 | 4E-07 | 3E-02 |
| | | Total (based on KM TEQs) (b) | 2E-06 | 1E-01 | 4E-07 | 3E-02 |
| (a) Cumulative cancer risks and hazard indices where TEQ is calculated manually.<br>(b) Cumulative cancer risks and hazard indices where TEQ is calculated using the KM TEQ calculator. | | | | | | |

*6.3.3.3 Wader — Adult*

The following table summarizes the cumulative potential cancer risks and noncancer HIs for the adult wader. The cumulative total potential cancer risks and noncancer hazards for this receptor are within or below the NCP risk range and below an HI of 1 (USEPA 1991d) for both the RME and CTE scenarios, regardless of TEQ approach.

| Wader<br>Adult | | | | | | |
|---|---|---|---|---|---|---|
| Media | RAGS D Table | Scenario | RME | | CTE | |
| | | | Total Risk | Total HI | Total Risk | Total HI |
| Accessible Surface Sediment | 9.5 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 1E-06 | 3E-02 | 2E-07 | 1E-02 |
| | | Total (based on KM TEQs) (b) | 1E-06 | 3E-02 | 2E-07 | 1E-02 |
| Surface Water | 9.5 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 1E-08 | 4E-04 | 1E-09 | 1E-04 |
| | | Total (based on KM TEQs) (b) | 1E-08 | 4E-04 | 1E-09 | 1E-04 |
| Baywide Cumulative Total Risk/Hazard | 9.5 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 1E-06 | 3E-02 | 2E-07 | 1E-02 |
| | | Total (based on KM TEQs) (b) | 1E-06 | 3E-02 | 2E-07 | 1E-02 |
| (a) Cumulative cancer risks and hazard indices where TEQ is calculated manually.<br>(b) Cumulative cancer risks and hazard indices where TEQ is calculated using the KM TEQ calculator. | | | | | | |

*6.3.3.4 Wader — Combined Adult/Child*

The following table summarizes the cumulative potential cancer risks the combined adult/child wader. The cumulative total potential cancer risks for this receptor are within or below the NCP risk range for both the RME and CTE scenarios, regardless of TEQ approach.

| **Wader**<br>**Combined Adult/Child (Cancer only) (a)** | | | | |
|---|---|---|---|---|
| Media | RAGS D Table | Scenario | RME Total Risk | CTE Total Risk |
| Accessible Surface Sediment | 9.B | Baywide | | |
| | | Total (excluding KM TEQs) (b) | 2E-06 | 3E-07 |
| | | Total (based on KM TEQs) (c) | 2E-06 | 3E-07 |
| Surface Water | 9.B | Baywide | | |
| | | Total (excluding KM TEQs (b) | 4E-08 | 6E-09 |
| | | Total (based on KM TEQs) (c) | 4E-08 | 6E-09 |
| Baywide Cumulative Total Risk/Hazard | 9.B | Mixed Fish Diet (a) | | |
| | | Total (excluding KM TEQs) (b) | 3E-06 | 4E-07 |
| | | Total (based on KM TEQs) (c) | 2E-06 | 3E-07 |

(a) Potential cancer risks in this table represent exposures for a child and adult over a 26-year period for RME and a 12-year exposure duration for CTE.

(b) Cumulative cancer risks where TEQ is calculated manually.

(c) Cumulative cancer risks where TEQ is calculated using the KM TEQ calculator.

6.3.4 Boater

Boaters are assumed to be exposed to COPCs in accessible surface sediment and surface water via dermal contact and incidental ingestion. Two age groups were evaluated: an adolescent (7 to 19 years), and an adult (>18 years). Young children (<7 years old) are not expected to participate in boating activities on the Bay; any such exposure would be rare and much less than that experienced by young children visiting the Bay specifically to wade or swim.

Potential cancer risks and noncancer hazards associated with adolescent and adult boater exposure to accessible surface sediment and surface water were estimated on a site-wide (or Bay-wide) basis. These values are summed to estimate cumulative cancer risks and noncancer hazards for these receptors. Cumulative sitewide cancer risks and noncancer hazards for the boater receptor are presented in Tables 6-1 and 6-2, respectively; results for each age group are discussed below.

*6.3.4.1 Boater – Adolescent*

The following table summarizes the cumulative potential cancer risks and noncancer HIs for the adolescent boater. The cumulative total potential cancer risks and noncancer hazards for this receptor are within or below the NCP risk range and below an HI of 1 (USEPA 1991d) for both the RME and CTE scenarios, regardless of TEQ approach.

| **Boater Adolescent (Age 7 to <19)** | | | | | | |
|---|---|---|---|---|---|---|
| Media | RAGS D Table | Scenario | RME | | CTE | |
| | | | Total Risk | Total HI | Total Risk | Total HI |
| Accessible Surface Sediment | 9.6 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 2E-06 | 1E-01 | 3E-07 | 3E-02 |
| | | Total (based on KM TEQs) (b) | 2E-06 | 1E-01 | 3E-07 | 3E-02 |
| Surface Water | 9.6 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 3E-07 | 9E-03 | 7E-08 | 5E-03 |
| | | Total (based on KM TEQs) (b) | 3E-07 | 9E-03 | 7E-08 | 5E-03 |
| Baywide Cumulative Total Risk/Hazard | 9.6 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 2E-06 | 1E-01 | 4E-07 | 4E-02 |
| | | Total (based on KM TEQs) (b) | 2E-06 | 1E-01 | 4E-07 | 4E-02 |

(a) Cumulative cancer risks and hazard indices where TEQ is calculated manually.
(b) Cumulative cancer risks and hazard indices where TEQ is calculated using the KM TEQ calculator.

*6.3.4.2 Boater — Adult*

The following table summarizes the cumulative potential cancer risks and noncancer HIs for the adult boater. The cumulative total potential cancer risks and noncancer hazards for this receptor are within or below the NCP risk range and below an HI of 1 (USEPA 1991d) for both the RME and CTE scenarios, regardless of TEQ approach.

| Boater<br>Adult | | | | | | |
|---|---|---|---|---|---|---|
| Media | RAGS D Table | Scenario | RME | | CTE | |
| | | | Total Risk | Total HI | Total Risk | Total HI |
| Accessible Surface Sediment | 9.5 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 4E-07 | 2E-02 | 6E-08 | 4E-03 |
| | | Total (based on KM TEQs) (b) | 4E-07 | 2E-02 | 6E-08 | 4E-03 |
| Surface Water | 9.5 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 3E-07 | 8E-03 | 4E-08 | 3E-03 |
| | | Total (based on KM TEQs) (b) | 3E-07 | 8E-03 | 4E-08 | 3E-03 |
| Baywide Cumulative Total Risk/Hazard | 9.5 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 7E-07 | 2E-02 | 1E-07 | 7E-03 |
| | | Total (based on KM TEQs) (b) | 7E-07 | 2E-02 | 1E-07 | 7E-03 |

(a) Cumulative cancer risks and hazard indices where TEQ is calculated manually.
(b) Cumulative cancer risks and hazard indices where TEQ is calculated using the KM TEQ calculator.

### 6.3.5 Worker

Workers are assumed to be exposed to COPCs in accessible surface sediment via dermal contact and incidental ingestion, which was estimated on a site-wide (or Bay-wide) basis. Sitewide cancer risks and noncancer hazards for the worker receptor are presented in Tables 6-1 and 6-2, respectively, which are summarized in the following table. The cumulative total potential cancer risks and noncancer hazards for this receptor are within or below the NCP risk range and below an HI of 1 (USEPA 1991d) for both the RME and CTE scenarios, regardless of TEQ approach.

| Worker<br>Adult | | | | | | |
|---|---|---|---|---|---|---|
| Media | RAGS D Table | Scenario | RME | | CTE | |
| | | | Total Risk | Total HI | Total Risk | Total HI |
| Accessible Surface Sediment | 9.4 | Baywide | | | | |
| | | Total (excluding KM TEQs) (a) | 3E-06 | 9E-02 | 3E-07 | 3E-02 |
| | | Total (based on KM TEQs) (b) | 3E-06 | 9E-02 | 3E-07 | 3E-02 |

(a) Cumulative cancer risks and hazard indices where TEQ is calculated manually.
(b) Cumulative cancer risks and hazard indices where TEQ is calculated using the KM TEQ calculator.

#### *6.3.5.1 Lead Risk Characterization*

Lead was identified as a COPC in accessible surface sediment and blue crab muscle/hepatopancreas tissue. The USEPA Integrated Exposure Uptake Biokinetic (IEUBK) model was used to quantify potential exposures to lead for children younger than 7 years of age (USEPA 1994a, 1994b). This model correlates lead levels in the environment to blood lead levels (PbB) in children. The model developed by Bowers et al.

(1994) was used to quantify exposures to lead for adolescent and adult receptors. The component of this model for soil is the same as that used in the USEPA Adult Lead Methodology (ALM) spreadsheet (USEPA 2017d). The ALM could not be used on its own because it does not account for exposure to lead via pathways other than soil (e.g., food). Adult and fetal PbBs were predicted using receptor-specific exposure parameters. For all receptors, PbBs were compared to 5 µg/dL, which is USEPA's target blood lead level used in the ALM spreadsheet (USEPA 2017d) and subsequent ALM guidance (USEPA 2017h), and consistent with CDC's current reference value for lead (CDC 2012). In addition, preliminary remediation goals (PRGs) were calculated for sediment and crab tissue using the same adult and adolescent-specific exposure parameters and a target PbB of 5 µg/dL. The results of the lead risk assessment are summarized below by receptor; the complete assessment is presented in Appendix E.

#### *6.3.5.2 Angler/Sportsman — Crab Consumption*

The predicted PbBs for children <7 years of age who may be exposed to lead via crab consumption are less than 5 µg/dL for >99.9% of the population. Similarly, for adolescents and adults who are exposed to lead via crab consumption and direct exposure to accessible surface sediment, the predicted PbBs are below 5 µg/dL and crab tissue and sediment concentrations are below the calculated PRGs. These results indicate that lead in crab tissue and accessible surface sediment does not represent a hazard to an angler/sportsman.

#### *6.3.5.3 Swimmers, Waders, and Boaters*

The predicted PbBs for children <7 years of age who may be exposed to lead via direct contact with accessible surface sediment while swimming or wading are less than 5 µg/dL for >99.8% of the population. The predicted PbBs for adolescents and adults who may be exposed to lead via direct contact with accessible surface sediment while swimming, wading, or boating are below 5 µg/dL and sediment concentrations are below the calculated PRGs. These results indicate that lead in accessible surface sediment does not represent a hazard to a recreational swimmer, wader, or boater.

#### *6.3.5.4 Workers*

The predicted PbB for adult workers who may be exposed to lead via direct contact with accessible surface sediment is below 5 µg/dL and sediment concentration is below the calculated PRG. These results indicate that lead in accessible surface sediment does not represent a hazard to an adult worker.

### 6.3.6 Risk Characterization Summary

The key findings of the risk characterization are summarized below and, with the exception of lead, in tables that follow.

Fish and Crab

Potential exposure to COPCs in a mixed fish diet (composed of equal parts American eel, bluefish, striped bass, summer flounder, and white perch) or a crab muscle and hepatopancreas diet by anglers/sportsmen may pose cancer risks greater than the NCP risk range under the RME scenario; however, risks are within the range under the CTE scenario. For noncancer effects, the estimated noncancer HIs associated with consumption of a mixed fish diet or crab muscle and hepatopancreas diet are greater than the goal of an HI of 1 (USEPA 1991d) under both the RME and CTE scenarios. For the RME scenario, which by definition represents a conservative exposure case that is above the average case but still within the range of possible exposures, the potential cancer risk to the combined adult/child angler/sportsman who routinely consumes self-caught fish or crab (34.6 g/day for an adult and 11.5 g/day for a child) or crab (21 g/day for an adult and 7 g/day for a child) over a period of 26 years is $8\times10^{-4}$ for either fish or crab ingestion, regardless of TEQ approach). For noncancer hazards, the RME HI for a child angler/sportsman, the most sensitive age group, is 40 for fish ingestion and 50 for crab ingestion, regardless of TEQ approach.

For the CTE scenario, which is based on average exposures to fish (3.9 g/day for an adult and 1.3 g/day for a child) or crab (3 g/day for an adult and 1 g/day for a child) over a period of 12 years, and which accounts for cooking loss for fish, the potential cancer risk for the combined adult/child sportsman is $3\times10^{-5}$ for fish ingestion and $5\times10^{-5}$ for crab ingestion, regardless of TEQ approach, which are within the NCP risk range of $10^{-6}$ to $10^{-4}$. The CTE noncancer HIs for a child angler/sportsman is 4 for fish and 7 for crab, regardless of TEQ approach. These values are approximately a factor of 10 lower than for the RME scenario, but still exceed an HI of 1 (USEPA 1991d).

The COPCs contributing most to the overall risk/hazard from fish or crab consumption are 2,3,7,8-TCDD, copper, PCB-126, and non-DL PCBs, and to a lesser extent, other PCDD/Fs and DL-PCBs, inorganics (arsenic for risk, methyl mercury for hazard) and pesticides (dieldrin for risk, 4,4'-DDD for hazards). Below is a summary of percent contributions of key COPCs (see Appendix H) for the RME scenario.[6] As noted above, these values are limited to fish or crab consumption only, because any additional potential risks from exposure to accessible surface sediment or surface water are 100-fold or lower.

Fish Consumption

- Potential cancer risk (combined adult/child scenario): 2,3,7,8-TCDD contributes approximately 28% (33% for all PCDD/Fs), PCB-126 contributes approximately 31% (39% for all DL-PCBs), and non-DL PCBs contributes approximately 18%, which equate to maximum risks of $2\times10^{-4}$ ($3\times10^{-4}$ for all PCDD/Fs), $3\times10^{-4}$ ($3\times10^{-4}$ for all DL-PCBs), and $2\times10^{-4}$, respectively. Minor contributors include

[6] Data presented for individual congeners are based on TEQs calculated manually; data presented for total PCDD/Fs and total DL-PCBs are based on TEQs calculated using the KM TEQ calculator. As discussed above and shown in multiple tables, the two approaches to TEQ estimation result in essentially the same overall risks/hazards.

pesticides (approximately 6%) and inorganic arsenic (approximately 4%), which equate to maximum risks of $4\times10^{-5}$ (maximum of $3\times10^{-5}$ for dieldrin) and $3\times10^{-5}$, respectively.

- Noncancer hazard (child scenario): 2,3,7,8-TCDD contributes approximately 19% (22% for all PCDD/Fs), PCB-126 contributes approximately 20% (26% for all DL-PCBs), and non-DL PCBs contribute approximately 32%), which equates to maximum HIs of 8 (10 for all PCDD/Fs), 9 (10 for all DL-PCBs), and 10 for non-DL PCBs. Minor contributors include pesticides (approximately 9%) and inorganics (approximately 10%), which equate to maximum HIs of 4 (maximum of 2 for 4,4'-DDD) and 4 (maximum of 2 for methyl mercury), respectively.

Crab Consumption

- Potential cancer risk (combined adult/child scenario): 2,3,7,8-TCDD contributes approximately 52% (60% for all PCDD/Fs), PCB-126 contributes approximately 19% (23% for all DL-PCBs), and non-DL PCBs contribute approximately 8%, which equate to maximum risks of $4\times10^{-4}$ ($5\times10^{-4}$ for all PCDD/Fs), $2\times10^{-4}$ ($2\times10^{-4}$ for all DL-PCBs), and $7\times10^{-5}$, respectively. Minor contributors include inorganic arsenic (approximately 6%) and pesticides (approximately 3%), which equate to maximum risks of $5\times10^{-5}$ and $2\times10^{-5}$ (maximum of $1\times10^{-5}$ for dieldrin), respectively.
- Noncancer hazards (child scenario): 2,3,7,8-TCDD contributes approximately 33% (38% for all PCDD/Fs), copper contributes approximately 26%, PCB-126 contributes approximately 12% (15% for all DL-PCBs), and non-DL PCBs contribute approximately 14%, which equates to maximum HIs of 20 (20 for all PCDD/Fs), 10, 6 (7 for all DL-PCBs), and 7, respectively. Minor contributors include inorganics other than copper (approximately 4%) and pesticides (approximately 3%), which equate to maximum HIs of 2 (maximum of 0.7 for methyl mercury) and 1 (maximum of 0.5 for 4,4'-DDD), respectively.

As discussed in Section 7.3.3, there is considerable uncertainty in the TEFs for DL compounds, particularly for some of the DL-PCBs. Consistent with USEPA (2010a), a sensitivity analysis was conducted to illustrate the impact of the TEFs on the overall risk estimates and percent contribution of individual congeners or groups of congeners. For all congeners except 2,3,7,8-TCDD, the lower- and upper-bound TEFs were the $10^{th}$ and $90^{th}$ percentiles from *in vitro* and *in vivo* studies included in the relative effects potency (ReP) database (USEPA 2010a). The TEF for 2,3,7,8-TCDD remains constant in all scenarios. Accordingly, while the estimated risk for 2,3,7,8-TCDD remains constant, the contribution to risk can change, as can the relative contribution of all PCDD/Fs, all DL-PCBs, and all PCBs (non-DL and DL-PCBs). For example, for the combined adult/child angler/sportsman who consumes a mixed fish diet, the percent contribution for 2,3,7,8-TCDD increases from 28% to 44% when using the lower-bound TEFs, but decreases to only 1% when using the upper-bound TEFs. Conversely, the percent contribution to overall risk for Total PCBs (DL-PCBs and Non-DL PCBs) increases from 37% when using lower-bound TEFs to 98% when upper-bound TEFs are used. Similarly, for crab muscle and hepatopancreas consumption, the percent contribution of 2,3,7,8-TCDD increases from 52% to 70% when using the lower-bound TEFs, but decreases to approximately 2% when using the upper-bound TEFs. The percent contribution to overall risk for Total PCBs (DL-PCBs and Non-DL PCBs) increases from 16% when using lower-bound TEFs to 96% when upper-bound TEFs are used (see Section 7.3.3).

Sediment and Surface Water

The estimated cancer risks associated with potential exposure to COPCs in accessible surface sediment and surface water while angling, swimming, wading, or boating are within or below the NCP risk range of $10^{-6}$ to $10^{-4}$ (maximum risk of $4\times10^{-6}$ for combined adult/child angler/sportsman). Similarly, the estimated noncancer HIs are below 1 (USEPA 1991d) (maximum HI of 0.2 for child swimmer and child wader).

Lead

No adverse health effects are expected to be associated with exposure to lead in crab tissue, or accessible surface sediment for any NBSA receptors.

| Receptor Population | Age Group | Summary of Receptor/Exposure Pathway Cancer Risks for NBSA Baseline Human Health Risk Assessment (a) Reasonable Maximum Exposure (RME) | | | |
|---|---|---|---|---|---|
| | | Accessible Surface Sediment | Surface Water | Mixed Fish Diet (b) | Crab Muscle & Hepatopancreas |
| Angler/Sportsman | Child | *Pathways Incomplete* | | 3E-04 | 3E-04 |
| | Adolescent | 2E-06 | 8E-08 | 3E-04 | 3E-04 |
| | Adult | 4E-06 | 5E-08 | 6E-04 | 6E-04 |
| | Adult/Child (c)l | 4E-06 | 5E-08 | 8E-04 | 8E-04 |
| Swimmer | Child | 1E-06 | 2E-07 | *Pathways Incomplete* | |
| | Adolescent | 2E-06 | 5E-07 | | |
| | Adult | 1E-06 | 9E-08 | | |
| | Adult/Child (c) | 2E-06 | 3E-07 | | |
| Wader | Child | 1E-06 | 3E-08 | | |
| | Adolescent | 2E-06 | 6E-08 | | |
| | Adult | 1E-06 | 1E-08 | | |
| | Adult/Child (c) | 2E-06 | 4E-08 | | |
| Boater | Child | *Pathways Incomplete* | | | |
| | Adolescent | 2E-06 | 3E-07 | | |
| | Adult | 4E-07 | 3E-07 | | |
| | Adult/Child (c) | *Not Applicable* | | | |
| Worker | Adult | 3E-06 | *Not quantified (d)* | | |

Notes:

Shading indicates that the cumulative potential carcinogenic risk exceeds 10-4.

(a) Cumulative cancer risks differ only minimally based on the method for estimating toxicity equivalency (TEQ) concentration (Kaplan-Meier [KM] TEQ calculator vs. manual calculations); therefore, the results presented represent the results based on the KM TEQ calculator.

(b) Mixed fish diet assumed to consist of equal fractions (20%) of American eel, bluefish, striped bass, summer flounder, and white perch.

(c) Cancer risks for adult and child age groups summed to yield 26-year total exposure duration.

(d) Workers are not expected to have contact with surface water during outdoor activities.

| Receptor Population | Age Group | Summary of Receptor/Exposure Pathway Cancer Risks for NBSA Baseline Human Health Risk Assessment (a) Central Tendency Exposure (CTE) | | | |
|---|---|---|---|---|---|
| | | Accessible Surface Sediment | Surface Water | Mixed Fish Diet (b) | Crab Muscle & Hepatopancreas |
| Angler/Sportsman | Child | *Pathways Incomplete* | | 9E-06 | 2E-05 |
| | Adolescent | 4E-07 | 8E-09 | 1E-05 | 2E-05 |
| | Adult | 6E-07 | 5E-09 | 2E-05 | 3E-05 |
| | Adult/Child (c) | 6E-07 | 5E-09 | 3E-05 | 5E-05 |
| Swimmer | Child | 2E-07 | 4E-08 | *Pathways Incomplete* | |
| | Adolescent | 3E-07 | 1E-07 | | |
| | Adult | 2E-07 | 2E-08 | | |
| | Adult/Child (c) | 3E-07 | 4E-08 | | |
| Wader | Child | 2E-07 | 1E-08 | | |
| | Adolescent | 3E-07 | 7E-09 | | |
| | Adult | 2E-07 | 1E-09 | | |
| | Adult/Child (c) | 3E-07 | 6E-09 | | |
| Boater | Child | *Pathways Incomplete* | | | |
| | Adolescent | 3E-07 | 7E-08 | | |
| | Adult | 6E-08 | 4E-08 | | |
| | Adult/Child (c) | *Not Applicable* | | | |
| Worker | Adult | 3E-07 | *Not quantified (d)* | | |

Notes:

Shading indicates that the cumulative potential carcinogenic risk exceeds 10-4.

(a) Cumulative cancer risks differ only minimally based on the method for estimating toxicity equivalency (TEQ) concentration (Kaplan-Meier [KM] TEQ calculator vs. manual calculations); therefore, the results presented represent the results based on the KM TEQ calculator.

(b) Mixed fish diet assumed to consist of equal fractions (20%) of American eel, bluefish, striped bass, summer flounder, and white perch

(c) Cancer risks for adult and child age groups summed to yield 12-year total exposure duration.

(d) Workers are not expected to have contact with surface water during outdoor activities.

| Receptor Population | Age Group | Summary of Receptor/Exposure Pathway Noncancer Hazards for NBSA Baseline Human Health Risk Assessment (a) Reasonable Maximum Exposure (RME) | | | |
|---|---|---|---|---|---|
| | | Accessible Surface Sediment | Surface Water | Mixed Fish Diet (b) | Crab Muscle & Hepatopancreas |
| Angler/Sportsman | Child | *Pathways Incomplete* | | 4E+01 | 5E+01 |
| | Adolescent | 1E-01 | 2E-03 | 3E+01 | 3E+01 |
| | Adult | 1E-01 | 1E-03 | 3E+01 | 3E+01 |
| Swimmer | Child | 2E-01 | 8E-03 | *Pathways Incomplete* | |
| | Adolescent | 1E-01 | 1E-02 | | |
| | Adult | 3E-02 | 3E-03 | | |
| Wader | Child | 2E-01 | 1E-03 | | |
| | Adolescent | 1E-01 | 2E-03 | | |
| | Adult | 3E-02 | 4E-04 | | |
| Boater | Child | *Pathways Incomplete* | | | |
| | Adolescent | 1E-01 | 9E-03 | | |
| | Adult | 2E-02 | 8E-03 | | |
| Worker | Adult | 9E-02 | *Not quantified (c)* | | |

Notes:

Total hazard index presented. Shading indicates that one or more target organ-specific hazard indices exceed one.

(a) Cumulative noncancer hazards differ only minimally based on the method for estimating toxicity equivalency (TEQ) concentration (Kaplan-Meier [KM] TEQ calculator vs. manual calculations); therefore, the results presented represent the results based on the KM TEQ calculator.

(b) Mixed fish diet assumed to consist of equal fractions (20%) of American eel, bluefish, striped bass, summer flounder, and white perch.

(c) Workers are not expected to have contact with surface water during outdoor activities.

| Receptor Population | Age Group | Summary of Receptor/Exposure Pathway Noncancer Hazards for NBSA Baseline Human Health Risk Assessment (a) Central Tendency Exposure (CTE) | | | |
|---|---|---|---|---|---|
| | | Accessible Surface Sediment | Surface Water | Mixed Fish Diet (b) | Crab Muscle & Hepatopancreas |
| Angler/Sportsman | Child | *Pathways Incomplete* | | 4E+00 | 7E+00 |
| | Adolescent | 4E-02 | 5E-04 | 2E+00 | 4E+00 |
| | Adult | 4E-02 | 4E-04 | 2E+00 | 4E+00 |
| Swimmer | Child | 5E-02 | 4E-03 | *Pathways Incomplete* | |
| | Adolescent | 3E-02 | 6E-03 | | |
| | Adult | 1E-02 | 1E-03 | | |
| Wader | Child | 5E-02 | 4E-04 | | |
| | Adolescent | 3E-02 | 4E-04 | | |
| | Adult | 1E-02 | 1E-04 | | |
| Boater | Child | *Pathways Incomplete* | | | |
| | Adolescent | 3E-02 | 5E-03 | | |
| | Adult | 4E-03 | 3E-03 | | |
| Worker | Adult | 3E-02 | *Not quantified (c)* | | |

Notes:

Total hazard index presented. Shading indicates that one or more target organ-specific hazard indices exceed one.

(a) Cumulative noncancer hazards differ only minimally based on the method for estimating toxicity equivalency (TEQ) concentration (Kaplan-Meier [KM] TEQ calculator vs. manual calculations); therefore, the results presented represent the results based on the KM TEQ calculator.

(b) Mixed fish diet assumed to consist of equal fractions (20%) of American eel, bluefish, striped bass, summer flounder, and white perch.

(c) Workers are not expected to have contact with surface water during outdoor activities.

### 6.4 Potential COC Identification

Potential COCs are identified for those scenarios where the estimated total potential cumulative cancer risk exceeds $10^{-4}$ or the noncancer HI exceeds one. It is important to note that potential COCs are identified for all media associated with a receptor population, even if the media-specific cancer risks or noncancer HIs do not exceed these criteria. For example, as discussed in Section 6.3.1.4, the estimated cumulative cancer risk for the combined adult/child angler/sportsman is greater than $10^{-4}$ based almost entirely on fish ingestion, with estimated risks associated with direct contact with sediment and surface water well within the risk range. Nevertheless, potential COCs are identified for all media.

Potential COCs are identified according to the following rules:

- When the receptor-specific estimated total potential cumulative cancer risk exceeds $10^{-4}$, any chemical with an exposure pathway-specific risk greater than $10^{-6}$ is a potential COC.
- When the receptor-specific estimated total potential cumulative target-organ-specific HI exceeds one, any chemicals with an exposure-pathway-specific, target-organ-specific HI greater than 0.1 is a potential COC.

Based on these rules, potential COCs are identified for fish and crab consumption and direct contact with accessible surface sediment, and these potential COCs are shown in the cumulative risk/hazard tables in RAGS Part D Table 10 format presented in Appendix I. Summary tables showing potential COCs for each receptor, age group, and scenario are provided in Tables 6-7 and 6-8, with chemicals divided into the following categories:

Cancer risks

- $>10^{-4}$
- $>10^{-5}$ and $\leq 10^{-4}$
- $>10^{-6}$ and $\leq 10^{-5}$

Noncancer HIs

- >1
- >0.1 and ≤1

As discussed previously, risk and hazard estimates for dioxin-like compounds are presented based on two approaches to calculating the TEQ, which are designated “Total PCDD/Fs (based on KM TEQ)” and “Total DL-PCBs (based on KM TEQ)” or “Total PCDD/Fs (excluding KM TEQ)” and Total DL-PCBs (excluding KM TEQ). Potential COCs could include individual congeners or Total PCDD/Fs and/or Total DL-PCBs, regardless of TEQ approach.

The primary chemicals/chemical groups driving the estimated total potential cumulative cancer risk and noncancer HI are 2,3,7,8-TCDD, PCB-126, and non-DL PCBs. For consumption of a mixed fish diet under the RME scenario, these chemicals collectively constitute approximately 78% of the total risk and approximately 73% of the total noncancer hazard for the RME angler/sportsman scenario. Total PCDD/Fs and Total DL-PCBs make up approximately 70% to 71% of the total potential cumulative cancer risk and 46% to 47% of the total noncancer hazard, depending on TEQ approach. For consumption of a crab muscle and hepatopancreas diet, 2,3,7,8-TCDD, PCB-126, and non-DL PCBs make up approximately 79% of both the total risk and hazard, with Total PCDD/Fs and Total DL-PCB constituting approximately 83% of the total potential cumulative cancer risk and 71% of the total noncancer hazard, regardless of TEQ approach.

Other than DL compounds and non-DL PCBs, various pesticides, inorganic arsenic, and methyl mercury contribute a few percent (6% or less) to the total cumulative cancer risk or noncancer hazard, although of these compounds, only methyl mercury and 4,4’-DDD were identified as potential COCs for the CTE scenario.

Potential COCs are summarized in Table 6-9 for both RME and CTE scenarios; potential COCs for the RME scenario are summarized by the medium and cancer risk/noncancer hazard range below. It should be noted that two chemicals identified as potential COCs in fish tissue—benzo(a)pyrene and dibenz(a,h)anthracene—were not actually detected in any of the fish tissue samples. This apparent anomaly occurred because, as noted in Section 3.4, “…for consistency, if a chemical was identified as a COPC in any fish or crab tissue, it

was retained as a COPC for all tissue types." These two chemicals were detected in crab tissue at concentrations exceeding their respective screening levels. The EPC used in the risk calculations was the maximum limit of detection (0.005 or 0.013 mg/kg, depending on fish species; see Table 4-17). As shown below, the estimated risks associated with these two chemicals fall between $10^{-6}$ and $10^{-5}$, and thus are minor contributors to the cumulative risk estimates.

| Identification of Potential Chemicals of Concern Based on RME Scenario (a) | | | | |
|---|---|---|---|---|
| Chemicals with Cancer Risk >$10^{-4}$ | Chemicals with Cancer Risk >$10^{-5}$ and ≤$10^{-4}$ | Chemicals with Cancer Risk >$10^{-6}$ and ≤$10^{-5}$ | Chemicals with Target Organ Effect HI>1 | Chemicals with Target Organ Effect HI>0.1 and ≤1 |
| **RME Mixed Fish Diet** | | | | |
| 2,3,7,8-TCDD | 1,2,3,7,8-PeCDD | 1,2,3,6,7,8-HxCDD | 2,3,7,8-TCDD | 1,2,3,7,8-PeCDD |
| PCB-126 | 2,3,4,7,8-PeCDF | 2,3,7,8-TCDF | PCB-126 | 2,3,7,8-TCDF |
| Total Non-DL PCBs | PCB-118 | 1,2,3,7,8-PeCDF | Total Non-DL PCBs | 2,3,4,7,8-PeCDF |
| Total PCDD/Fs (excluding KM TEQ) | Dieldrin | 1,2,3,4,7,8-HxCDF | 4,4'-DDD | 1,2,3,4,7,8-HxCDF |
| Total PCDD/Fs (based on KM TEQ) | Arsenic, inorganic | 1,2,3,6,7,8-HxCDF | Methyl Mercury | PCB-105 |
| Total DL-PCBs (excluding KM TEQ) | | PCB-77 | Total PCDD/Fs (excluding KM TEQ) | PCB-118 |
| Total DL-PCBs (based on KM TEQ) | | PCB-105 | Total PCDD/Fs (based on KM TEQ) | PCB-169 |
| | | PCB-156/157 | Total DL-PCBs (excluding KM TEQ) | 2,4'-DDD |
| | | PCB-167 | Total DL-PCBs (based on KM TEQ) | 4,4'-DDE |
| | | PCB-169 | | Dieldrin |
| | | Benzo(a)pyrene | | Nonachlor, trans- |
| | | Dibenz(a,h)anthracene | | Pyridine |
| | | 4,4'-DDD | | Arsenic, inorganic |
| | | 4,4'-DDE | | Cobalt |
| | | Chlordane, alpha (cis) | | Copper |
| | | Heptachlor epoxide, cis- | | Mercury |
| **Crab Muscle & Hepatopancreas** | | | | |
| 2,3,7,8-TCDD | 1,2,3,7,8-PeCDD | 1,2,3,7,8-PeCDF | 2,3,7,8-TCDD | 1,2,3,7,8-PeCDD |
| PCB-126 | 2,3,7,8-TCDF | 1,2,3,4,7,8-HxCDF | PCB-126 | 2,3,7,8-TCDF |
| Total PCDD/Fs (excluding KM TEQ) | 2,3,4,7,8-PeCDF | 1,2,3,6,7,8-HxCDF | Total Non-DL PCBs | 2,3,4,7,8-PeCDF |
| Total PCDD/Fs (based on KM TEQ) | PCB-118 | PCB-77 | Total PCDD/Fs (excluding KM TEQ) | 1,2,3,4,7,8-HxCDF |
| Total DL-PCBs (excluding KM TEQ) | Total Non-DL PCBs | PCB-105 | Total PCDD/Fs (based on KM TEQ) | PCB-77 |
| Total DL-PCBs (based on KM TEQ) | Dieldrin | PCB-156/157 | Total DL-PCBs (excluding KM TEQ) | PCB-105 |
| | Arsenic, inorganic | PCB-169 | Total DL-PCBs (based on KM TEQ) | PCB-118 |
| | | 4,4'-DDE | | PCB-169 |
| | | Heptachlor epoxide, cis- | | 4,4'-DDD |
| | | Heptachlor epoxide, trans- | | 4,4'-DDE |
| | | | | Heptachlor epoxide, cis- |

| Identification of Potential Chemicals of Concern Based on RME Scenario (a) | | | | |
|---|---|---|---|---|
| Chemicals with Cancer Risk >$10^{-4}$ | Chemicals with Cancer Risk >$10^{-5}$ and ≤$10^{-4}$ | Chemicals with Cancer Risk >$10^{-6}$ and ≤$10^{-5}$ | Chemicals with Target Organ Effect HI>1 | Chemicals with Target Organ Effect HI>0.1 and ≤1 |
| | | | | Nonachlor, trans-<br>Pyridine<br>Arsenic, inorganic<br>Cadmium<br>Cobalt<br>Copper<br>Mercury<br>Methyl Mercury |
| **Accessible Surface Sediment** | | | | |
| None | None | Arsenic, inorganic | None | None |
| **Surface Water** | | | | |
| None | None | None | None | None |

(a) The potential COCs for cancer risk are based on combined adult/child angler/sportsman receptor. The potential COCs for noncancer hazard are based on child angler/sportsman receptor.

# 7. Uncertainty Evaluation

The risk assessment process requires assumptions to be made about conceptual models and quantitative factors that affect the resulting risk characterization. These assumptions are made in the presence of variability (e.g., different body weights) and uncertainty (e.g., imperfect knowledge about toxicity). The risk assessment includes considerations of the variability and uncertainty in the exposure assumptions, to ensure the risks are not underestimated.

According to USEPA (https://www.epa.gov/expobox/uncertainty-and-variability):

> Variability *refers to the inherent heterogeneity or diversity of data in an assessment. It is a quantitative description of the range or spread of a set of values and is often expressed through statistical metrics such as variance, standard deviation, and interquartile ranges that reflect the variability of the data.*
>
> Uncertainty *refers to a lack of data or an incomplete understanding of the context of the risk assessment decision. It can be either qualitative or quantitative.*

Qualitative uncertainty may be due to a lack of knowledge about the factors that affect risk, whereas quantitative uncertainty may come from non-precise measurement methods or limited available studies.

Variability cannot be reduced, but it is addressed in deterministic risk assessments by using a combination of assumed values for variable parameters that estimates “average” exposure (i.e., CTE) and “high-end” exposure (i.e., RME). Uncertainty can be reduced with more study and data, but scientific, economic, and time constraints can limit the level of reduction that is practically feasible.

The following sections present an evaluation of variability and uncertainty inherent to the BHHRA. Section 7.1 addresses variability and uncertainty in data evaluation and potential COC selection, Section 7.2 addresses variability and uncertainty in exposure assessment, Section 7.3 addresses variability and uncertainty in toxicity assessment, and Section 7.4 addresses variability and uncertainty in risk characterization.

## 7.1 Data Evaluation and Potential COC Selection

### 7.1.1 Adequacy and Quality of Analytical Data

The physical, chemical, and biological data collected as part of the RI/FS programs conducted for the NBSA (sediment and biota) and LPRSA (surface water) between 2011 and 2014 serve as the foundation of the BHHRA. These data were collected according to USEPA Region 2–approved sampling plans and QAPPs developed based on an evolving conceptual model for the site, including site conditions and the fate and transport characteristics of chemicals found in the NBSA. The CSM was refined as new information became available, which served to guide subsequent sampling programs. The combined chemistry and survey data (e.g., fish community) provide a high level of confidence that the environmental conditions and range of

impacts within the NBSA have been sufficiently characterized for purposes of the BHHRA. As agreed with USEPA, the accessible surface sediment samples were limited to those so identified in Table 2 of the SQT QAPP (Tierra, 2015b), surface water samples were limited to those collected from six locations in the NBSA from the surface to a depth of approximately 3 feet (AECOM 2019), fish-tissue samples were from sampling programs in 2014, 2015, and 2016 (Tierra 2017), and crab tissue samples were from the Crab and Clam program (GSH 2017a). Sediment, surface water and/or tissue samples were analyzed for a wide variety of compounds, including PCDD/Fs, PCBs, PAHs, SVOCs, VOCs, pesticides, herbicides, and inorganics, although not every sample was analyzed for all chemicals. While it is possible that chemicals not included in these analytical suites may be present in environmental media at the NBSA, the comprehensive nature of the analytical program included the chemicals used by industry in the NBSA and industry in general. Accordingly, the chemicals of potential public health concern that are associated with the NBSA are included in the data set underpinning the BHHRA.

All of the laboratory results for sediment, surface water, and tissue considered for use in the BHHRA underwent formal data validation. Only a small fraction (2% in sediment, 0.3% in surface water, and 2% in biota) were determined to be invalid (R-qualified), and the remaining data were used in the BHHRA as reported, either unqualified or qualified (J- or U-qualified). Data usability worksheets in RAGS Part D format are provided in Attachments A-1 through A-4 to Appendix A, which summarize the results of the data validation process and provide a brief summary of the analysis and conclusions. Additional information can be found in specific field data reports. While inclusion of J-qualified data adds some uncertainty, because the true concentration is unknown, the use of these data in the BHHRA is consistent with USEPA (1989) guidance.

### 7.1.2 Adequacy of the COPC Selection Process

The COPC screening process is intended to identify the chemicals that require further evaluation in the BHHRA, because they can potentially cause adverse health effects in humans exposed at the site. As discussed in Section 3.3, COPCs were identified through (1) identification of compounds classified by USEPA as a known human carcinogen, (2) evaluation of detection frequency, (3) identification of essential nutrients, and (4) comparison of the maximum concentration to risk-based screening values. A summary of the screening process is provided in Figure 3-7.

Chemicals identified as COPCs in the BHHRA were detected at least once, met a minimum detection frequency requirement of 5%, and also had a maximum concentration in the exposure media that exceeded its risk-based toxicity screening level. All detected known human carcinogens (as classified by USEPA [2019b, 2019d, 2005b]) were retained in the quantitative risk assessment as COPCs regardless if they met the minimum detection frequency requirement of 5%, and regardless of reported concentrations in the exposure media. Other chemicals were flagged in the COPC process as requiring a qualitative evaluation of uncertainty, but were excluded from the quantitative risk evaluation, for any of the following reasons:

1. The chemical was not detected in any of the samples and was classified by USEPA as a known human carcinogen, or the chemical was not detected and its maximum detection limit (DL) exceeded the risk-based screening level.

2. The chemical was detected in less than 5% of samples, and its maximum concentration (either detected concentration or a non-detect proxy value) exceeded the risk-based screening level.
3. A risk-based screening value could not be identified for the chemical, and a suitable surrogate could not be identified.

The uncertainty associated with excluding these chemicals from the quantitative risk assessment is discussed herein and examined in Tables B-1.2 and B-1.3 in Appendix B-1.

*7.1.2.1 Chemicals excluded from quantitative risk assessment — Not detected*

Chemicals that were not detected in a given medium were eliminated from the quantitative risk assessment and flagged for uncertainty review. If a chemical's analytical method is not sensitive enough to measure concentrations within the range of the risk-based screening level, it is possible that excluding these chemicals based on zero detection can underestimate potential risks. To address this uncertainty, detection limits for non-detected chemicals in crab, fish, sediment, and surface water were compared to their corresponding risk-based screening level. Table B-1.2 in Appendix B-1 presents each of the chemicals flagged for uncertainty review, along with their minimum/maximum detect limits (DLs) and screening levels (SLs). Additionally, Table B-1.3 presents the unique list of chemicals across all media and identifies with an asterisk (*) if the chemical was flagged for uncertainty review in a given medium (i.e., crab, fish, sediment, or surface water). Further, if the chemical was flagged in the particular medium, the ratio between the minimum DL and SL is presented.

Of the 56 chemicals requiring uncertainty review, 45 chemicals were flagged for not being detected in one of the exposure media (see Table B-1.3). Two chemicals, 4-bromophenyl phenyl ether and 4-chlorophenyl phenyl ether, were not detected in any of the media and did not have risk-based screening levels. Detection levels were identical for these compounds and ranged as follows: sediment, 0.021–0.043 mg/kg; surface water, 0.94–1.1 µg/L; biota, 0.32–0.33 mg/kg. Given the lack of both toxicological reference values and detections in the various exposure media, it is assumed that these chemicals present minimal risk for exposed receptors in the NBSA.

The known human carcinogens that were not detected in one or more media include benzene, benzidine, and vinyl chloride. The detection limits and screening levels for benzene and vinyl chloride (analyzed in surface water) are within similar ranges and are not anticipated to contribute significantly to total risk. A review of the DLs and SLs for benzidine indicates that its analytical sensitivity is not adequate in sediment and biota tissues, with its SL being three to five orders of magnitude lower than the minimum detection limits. If benzidine was actually present at a level between the DL and the SL, it may present a non-negligible risk, and overall risk may be underestimated.

For the remaining 40 chemicals, the ratio between the minimum DL and SL was reviewed. A majority of these chemicals had minimum DLs within two orders of magnitude of the screening level (i.e., minimum DL:SL ratio of less than 100). There were three exceptions:

- 1,2-dibromo-3-chloropropane

- 1,2-dibromoethane
- n-nitroso-di-n-propylamine.

Among these chemicals, 1,2-dibromo-3-chloropropane and 1,2-dibromoethane were analyzed only in surface water, and SLs are approximately three and two orders of magnitude lower than the minimum DL, respectively. Because these compounds were not detected, and the surface water risk-based screening level was based conservatively on residential exposure assumptions (as opposed to the actual risk of infrequent exposure to surface water during recreational activities), it is unlikely that the decision to exclude these chemicals will affect the results of the BHHRA. The remaining organic with greater than two orders of magnitude between the DL and SL (n-nitroso-di-n-propylamine) was flagged for biota (crab and/or fish) samples. This compound was not identified as a COPC in any other media (surface water or accessible sediment), as indicated in Table 3-13.

It should be noted that, while benzo(a)pyrene, dibenz(a,h)anthracene, and pyridine were flagged for uncertainty review, these compounds were actually evaluated quantitatively in the risk assessment, because they were selected as COPCs in one of the other biota matrices. As discussed in Section 6.4, benzo(a)pyrene and dibenz(a,h)anthracene were identified as potential COCs in fish tissue even though neither chemical was detected in any fish tissue samples. This apparent anomaly occurred because these chemicals were identified as COPCs in crab tissue and were thus retained as a COPC for all tissue types. The estimated risks associated with these two chemicals, based on an EPC equal to the maximum limit of detection, fall between $10^{-6}$ and $10^{-5}$, and thus are minor contributors to the cumulative risk estimates.

#### *7.1.2.2 Chemicals excluded from quantitative risk assessment — Detected*

Of the 56 chemicals requiring uncertainty review, 11 chemicals were detected in at least one exposure medium. Two of these chemicals, sulfide and titanium, did not have risk-based screening levels. Sulfides were detected in 100% of sediment samples; however, no further information was available regarding the speciation of sulfide represented in the analytical data. Titanium was not detected in four of the five fish species (i.e., it was detected with a frequency of 10% in striped bass), and detection limits ranged from 0.16 to 3.7 mg/kg in fish tissue. Additionally, titanium was detected in 74% of crab hepatopancreas samples and 22% of crab muscle samples. Titanium is a naturally occurring metal, and humans are exposed routinely through food and consumer products. The uncertainties associated with excluding sulfides and titanium are not expected to influence the overall risks estimated in the BHHRA.

The remaining compounds had low detection frequencies (between 0 and 5%), but the maximum chemical concentration (either a detected value or a non-detect proxy) exceeded its screening level:

- 1,2-diphenylhydrazine
- 1,2,4-trichlorobenzene
- 1,4-dichlorobenzene
- 2,4-dinitrotoluene
- 3,3'-dichlorobenzidine
- antimony

- benzaldehyde
- benzo(j,k)fluoranthene
- cyanide.

The maximum concentrations for these chemicals that were used in the screening evaluation (to compare to their corresponding risk-based screening levels) were actually non-detect proxy values instead of detected concentrations. This is true for all of the chemicals listed above, except antimony. Additionally, none of the chemicals listed above had minimum DL/SL ratios that exceeded two orders of magnitude, suggesting that the analytical detection capability was sufficient for these chemicals in the various media. Given the low detection frequency and detection limits within two orders of magnitude of the screening level, it is unlikely that the decision to exclude these chemicals will affect the results of the BHHRA.

It should be noted that benzaldehyde was flagged for uncertainty due to not being detected in any of the five species of fish, and having low detection in crab muscle tissue, while having a maximum concentration that exceeded an SL. However, benzaldehyde was identified as a COPC for crab hepatopancreas tissue and consequently was included in the quantitative risk assessment for all biota tissues. Benzaldehyde was not identified as a potential COC in either crab or fish tissue.

#### *7.1.2.3 Lead*

As discussed in Appendix E, potential exposure to lead in crab was evaluated based on the mean concentration of lead in hepatopancreas tissue (0.411 mg/kg) because this was the only tissue in which the maximum lead concentration exceeded the screening level. Had the assessment been done on the mean concentration in hepatopancreas and muscle combined (0.0553 mg/kg) or muscle only (0.0207 mg/kg), the estimated blood lead concentrations would have been even lower.

### 7.2 Exposure Assessment

Variability is inherent to exposure assessment, because humans vary in characteristics (e.g., body weight), behaviors (e.g., frequency of activities), and location during activities (exposure points), all of which affect their exposure.

Variability in exposure assessment is evaluated below by identifying variable parameters and quantifying their variability, explaining how a combination of assumed values for variable parameters estimates “average” exposure (i.e., CTE) and “high-end” exposure (i.e., RME), and analyzing the sensitivity of the BHHRA to variability in exposure estimation parameters.

Uncertainty in exposure assessment can be introduced by:

- Judgements, such as deciding to exclude pathways as likely to be incomplete or insignificant or to exclude receptor populations as being absent or not significantly exposed to potential COCs associated with site media

- Assumed characteristics of receptors, such as involvement in activities and amount of skin exposed during certain activities
- Data gaps and random errors in measurement or sampling techniques used to develop exposure point concentrations
- Assumed relationships within models used for exposure assessment—for example, additivity or linearity.

Uncertainty in the exposure assessment is evaluated below by qualitative discussion that presents the sources of uncertainty, identifies data gaps, explains any subjective decisions or instances where professional judgment was used, and discusses the likely impact of uncertainties regarding under- or over-estimation of risk. While elements of this discussion may be applicable to both the CTE and RME scenarios, risk management decisions are based on the RME scenario (USEPA 1989).

### 7.2.1 Exposure Pathway and Receptor Selection

As discussed in Section 4.1, some exposure pathways and receptors are not included in the quantitative estimation of risks. These include inhalation of volatile and semivolatile organic COPCs in sediment and surface water, ingestion of waterfowl or species other than fish and crabs, residential receptors, and transient receptors. Note that the inhalation pathways are given quantitative analysis, as described in the paragraph below, but are not added into the final cumulative risks of the BHHRA.

In Appendix C-1, a screening assessment for the inhalation of volatile and semivolatile COPCs from exposed NBSA sediments was conducted to determine whether this route of exposure should be included in the BHHRA. Calculated screening levels were compared to upper-bound Newark Bay sediment concentrations to determine whether potentially elevated carcinogenic risk and noncarcinogenic hazards are present. Based on these results, the sediment volatilization pathway was excluded from the BHHRA. Similarly, in Appendix C-2, the potential for exposure to volatile or semivolatile organic COPCs in surface water via inhalation of vapors in ambient air was evaluated. As shown in the appendix, the estimated annual average air concentrations for all four COPCs were below their respective residential air RSLs, by at least an order of magnitude. Accordingly, the surface water volatilization pathway was not included in the quantitative risk assessment. These screening calculations demonstrate that exclusion of these potentially complete inhalation pathways will not result in significant underestimation of exposures and risks in the BHHRA.

Exposure by ingestion of waterfowl or species other than fish and crabs (e.g., turtles, frogs) is not included in the quantitative risk assessment calculations. The New Jersey Division of Fish and Wildlife, Bureau of Law Enforcement has not observed anyone hunting in the NBSA (USEPA 2017a). Ingestion of waterfowl and animals other than fish/crabs at the NBSA appears to be minimal, especially relative to fish and crab consumption. Ducks are fattier than fish, and crabs and may carry a higher burden of PCBs/TCDD in their tissue. However, the types of waterfowl observed in the NBSA consume grass, not fish, which results in lower tissue concentrations. While there is anecdotal evidence of catching and eating turtles (AECOM 2017), it is improbable that BHHRA risks are underestimated by not including consumption of species other than fish and crab, because the likely frequency and amount of consumption is significantly lower than for fish and crab.

Residential receptors are not included as an exposed population in the quantitative risk assessment calculations. The Newark Bay shoreline does not appear to support residential land use, because, although there are residences near the Bay, access to the Bay from the residential properties is limited by physical barriers such as steep slopes and rocks. Limited residential areas were observed along the eastern shore of the Bay; these areas have either man-made or natural barriers to impede human access to the Bay. Surface water from the Bay is not used as a domestic water supply, although it is possible that residents could contact surface water during activities near their homes. It is also expected that the contribution from such exposures would be insignificant compared to the recreational pathway exposures included in the BHHRA. As such, excluding residential use exposures will not affect confidence that the estimates from the quantitative BHHRA are RME.

There are observations that transient persons inhabit areas near and along the NBSA and may be exposed to NBSA sediment and surface water via incidental ingestion and dermal contact. Exposure factors for this population are not currently available and it is difficult to collect this type of information within the time frame of this decision. While it is possible for short-term exposures to these receptors, the assumed exposures by long-term recreators and anglers/sportsmen (sediment, surface water, fish/crab consumption) are anticipated to be higher than those of a transient individual. Therefore, it is unlikely that excluding the transient receptor from the quantitative assessment will result in underestimating exposure and risks associated with the NBSA.

### 7.2.2 Exposure Scenario Assumptions

Exposure scenarios in the BHHRA involve exposure—by fishing, crabbing, eating fish and crab, wading, swimming, boating, and working—to COPCs in surface water, sediment, and fish/crab tissue. Assessing the various exposure scenarios associated with adults and children engaged in these activities requires making assumptions about the parameters affecting such exposures; for example, frequency and time in contact with media containing COPCs, rates of ingestion, body weight, skin surface area exposed, dermal adherence, and bioavailability of the COPC in the medium to which the person is exposed.

The exposure parameters have variability and uncertainty, because:

- Humans are different in age, gender, body, and behavior
- Characteristics and attributes of the location of exposure affect activities
- Information gaps exist with regard to chemical-specific factors such as dermal absorption fractions and oral bioavailability.

One combination of assumptions evaluated is meant to be representative of RME that is above the average case but within the range of reasonably possible exposures (USEPA 1992a). Exposure parameters that are variable or uncertain are selected to be a mix of average and higher-end values within their ranges, avoiding the unrealistically high exposure estimate that would result from using all upper-bound or maximum values. Another combination is meant to be representative of CTE that is the average level of exposure predicted for the receptors (USEPA 1992a). This estimate is developed by assuming average or central tendency values for most or all exposure assumptions.

A number of the values used in the BHHRA are standard default exposure parameter assumptions recommended by USEPA for Superfund site human health risk assessments (USEPA 1989, 1991b, 2004b, 2011, 2012c, 2014). These default assumptions for the RME scenarios were developed by USEPA, in many cases based on large amounts of data from the U.S. population (e.g., body weight and body part skin surface area), to be used in combination to represent a person (within certain age groups) experiencing the upper range of possible exposures. Use of these national default values accounts for variability and contributes little if any uncertainty to RME exposure estimates in the BHHRA.

For some parameters in the BHHRA, adjustments to the USEPA default values are made to account for differences in applicable media (e.g., soil versus sediment), activity (e.g., wading versus reed-gathering), or CTE estimation. Finally, some exposure parameters are based on site-specific or chemical-specific information. The effects of these assumptions on uncertainty about exposure estimates calculated in the BHHRA are discussed in the following subsections.

#### *7.2.2.1 Sediment and Surface Water Exposures*

Sediment and surface water exposures at the NBSA are associated with recreational uses of the Bay. A number of attributes of the NBSA make it a less than desirable place to visit for recreational purposes. These include access limitations from the shoreline types (i.e., bulkhead, bridges, sheet piling, and mudflats), poor water quality, limited abundance of target species for anglers, fish and crab consumption advisories and bans, urban/industrial/commercial setting, and limited availability of boat launches and beaches. Also, exposure frequencies for swimming are from estimates developed based on swimming pools, which are likely to be higher than for swimming in the Bay. In addition, all of the receptor's body surface area is assumed to be exposed to surface water for the entire exposure. This is unlikely, even for swimming, because exposure is probably more intermittent. Furthermore, exposure assumptions for the adolescent boater were developed for the LPRSA (AECOM 2017) and are based on assumed involvement in organized rowing. It is expected that such rowing is unlikely to occur in Newark Bay; therefore, these assumed values overestimate exposure frequency for the adolescent boating scenario at the NBSA.

For all of these reasons, the use of default assumptions for RME exposure frequencies in the BHHRA leads to overestimation of potential exposure to surface water and sediment by NBSA recreators. Even with these conservative assumptions, the risks estimated by the BHHRA from exposure to surface water and sediment during swimming, wading, boating, and angling are minor contributors to total risks (see Section 6).

Sediment Ingestion Rate

The sediment ingestion rates assumed (50 mg/day for adults and adolescents and 100 mg/day for children) in the RME scenarios are 50% of the USEPA default values for soil ingestion. It is expected that some level of sediment removal by surface water will result in less hand-to-mouth loading than is the case with soil ingestion. Also, the USEPA soil ingestion rates represent the total daily intake of soil integrated over a variety of activities and sources, both indoors and outdoors (home, work, school, etc.). Furthermore, results of more recent studies (Stanek et al. 1997, 1999, Stanek and Calabrese 2000) have been published by the same investigators as the original studies upon which USEPA's default soil ingestion rates are based. The

more recent studies incorporate improvements in study design and analysis and address some of the issues and uncertainties associated with the earlier studies. These studies suggest that upper-bound estimates of long-term soil ingestion for children and adults are approximately half of the older estimates (or 100 and 50 mg/day, respectively), and central tendency estimates are approximately one fifth of the older CTE values (or 20 and 10 mg/day, respectively). Therefore, while there is uncertainty about the soil ingestion rates that best represent sediment ingestion at the NBSA, the rates assumed are likely to represent high-end estimates and not underestimate corresponding exposures and risks.

Sediment-on-Skin Adherence Factor

The Exposure Factors Handbook (USEPA 2011a) recommends soil/sediment adherence factors for adolescents and children based on a study of children engaged in shoreline play on tidal flats. However, review of the original study upon which this USEPA recommendation is based (Shoaf et al. 2005) revealed that the sediment in the study had a larger grain size than is typically found in the sediment associated with the NBSA. The adherence of sandy sediment, as characterized in the Shoaf et al. (2005) study, may be less than adherence of finer-grain sediment. The activity and conditions that most reasonably compare with child receptor activities involving exposure of skin to sediment at the NBSA, for which there are available adherence estimates (USEPA 2011a), is assumed to be children playing in wet soil. These values will not underestimate adherence.

The adherence factor of 0.3 mg/cm$^2$ for adults is based on the geometric mean of the reed gatherer population from Exhibit 3-3 of RAGS Part E (USEPA 2004b) and is a weighted adherence factor based on hands, lower legs, forearms, and feet. This assumed adherence factor is a reasonable assumption for evaluating dermal exposure to NBSA sediment during recreational and worker activities, because these activities all involve exposure of similar body parts and movements. This assumption does not underestimate exposure or risks corresponding to this pathway and route.

Surface Water Exposure Assumptions

Exposure frequencies for RME surface water contact scenarios range from 13 to 39 days per year for the angler/sportsman, swimmer, and wader, whereas the RME exposure frequencies for the boater are 98 days per year for the adolescent and 259 days per year for the adult. Exposure times to surface water for the RME scenario are 1 hour per day for the angler/sportsman and wader, 2 hours per day for the boater, and 2.6 hours per day for the swimmer. Exposure times and frequencies for the CTE scenarios are one-half to three-quarters of the RME scenario values. There is relatively little information available regarding these types of activities; therefore, many of these assumptions are based on professional judgment and are therefore inherently uncertain. Exceptions include the exposure time for swimmers, which is based on a reported national average for swimming (USEPA 1989), and the exposure frequency for adolescent and adult boaters, which is based on assumed involvement in organized rowing to be consistent with the LPRSA BHHRA (AECOM 2017). Given the limited points of access to Newark Bay, the lack of designated swimming areas, and visible deterrents such as trash and debris, it is likely that the RME and CTE exposure assumptions overestimate exposure from swimming, and particularly for boaters, where organized rowing would not be expected to occur. Finally, the exposed skin surface area is assumed to be in contact with

surface water for the entire exposure time, which is likely an overestimate for anglers/sportsmen and boaters, because surface water contact would be expected to be intermittent. Nevertheless, the estimated cancer risks and noncancer hazards associated with direct contact with surface water are well below the NCP risk range and noncancer protection goal of 1 (USEPA 1991d).

#### *7.2.2.2 Fish and Crab Consumption Exposures*

The most significant pathway by which people may be exposed to chemicals in the NBSA is from consuming contaminated fish and/or shellfish. A number of attributes of the NBSA make it a less-than-desirable place to visit for recreational purposes. These include access limitations from the shoreline (i.e., bulkhead, bridges, sheet piling, and mudflats), poor water quality, limited abundance of target species for anglers, fish and crab consumption advisories and bans, and urban/industrial/commercial setting. Therefore, the use of default exposure assumptions may not best represent anglers in the NBSA. Site-specific assumptions for the following parameters have been used to assess CTE and RME risks for fish and crab: fish consumption rate, crab consumption rate, and fraction ingested from contaminated source (fish/crab). The uncertainties associated with site-specific fish and crab exposure assumptions are discussed below.

Fish consumption

Fish ingestion rates used in the BHHRA were developed by USEPA Region 2 as part of the LPRSA BHHRA (USEPA 2012a). To derive their recommended RME and CTE values for adult anglers (34.6 and 3.9 g/day, respectively), USEPA averaged the 90$^{th}$ and 50$^{th}$ percentiles reported by two surveys: an intercept survey of the Newark Bay Complex (Burger 2002) and a mail survey of licensed anglers in the State of New York (Connelly et al. 1992). Fish ingestion rates for the child and adolescent receptors were estimated, assuming rates that are one-third and two-thirds of the adult ingestion rates, respectively.

The Newark Bay 1999 angler survey (Burger 2002) intercepted anglers that fished and/or crabbed at various locations in the Newark Bay Complex, including Newark Bay, Hackensack River, Passaic River, Arthur Kill, and Kill van Kull. The survey was conducted by researchers at Rutgers University, and sites were visited randomly between May and September of 1999 throughout the day on weekdays and weekends. Survey respondents provided information on their fish and/or crab consumption behavior, reasons for angling, knowledge of advisories, and demographics. Of the 267 anglers that were interviewed in the survey, 65 responded that they only fish (i.e., they do not go crabbing) and they eat their self-caught fish.

Connelly et al. (1992) surveyed licensed anglers in the State of New York via mail. Of the 2000 mail questionnaires sent to anglers, 1030 were completed and returned. Of the non-respondents, 100 were contacted directly via phone to address non-response bias. USEPA reviewed data for the 226 survey respondents who reported consuming fish from flowing waters. Additionally, 55 of the non-respondents were included as they reported consuming at least one or more meals from their catch.

The following are uncertainties associated with the fish ingestion rate:

- *Portion Size* — The Burger (2002) survey relied on a visual model to estimate portion sizes. Subjects were provided with a 3-dimensional model of an 8-oz fillet of fish and asked to estimate their average meal portion size. This reported portion size was assumed to be the same for all meals throughout the year. USEPA's reanalysis of this data set identified four records of unusually large portion sizes of greater than 30 ounces (or greater than 2 pounds). These records were excluded from the analysis. The Connelly et al. (1992) survey only collected the number of self-caught meals consumed by the angler and did not collect data on portion size. To compute an ingestion rate, the portion size of every meal was assumed to be 8 ounces. These uncertainties may over- or under- estimate consumption.
- *Recall Bias* — Uncertainty is associated with using single recall survey events (e.g., mail surveys or creel surveys) to estimate long-term consumption rates, because it is difficult for participants to remember their activities over an entire year. When asked to recall activities over the past year, mail-recall survey respondents tend to overestimate their activities, particularly in the case of more frequent anglers (USEPA and USACE 2000). Further, conducting the creel survey during the peak fishing season (as in the case of Burger 2002), can result in an overestimation of catch throughout the year and subsequently an overestimation of consumption.
- *Response Rate* — Connelly et al. (1992) had a survey response rate of 52.3%. Low response rates typically bias consumption estimates toward higher consumers, because non-respondents usually consume less then individuals who do respond. According to Table D-1 of Connelly et al. (1992), the average number of self-caught meals reported by mail respondents was 20.4, whereas the average number of self-caught meals reported by a subset of non-respondents (later contacted via phone) was 7.6 (Connelly et al. 1992). This uncertainty can lead to an overestimation of consumption.
- *Species-specific ingestion rates* — Neither of the data sets provided insight into the particular species of fish caught and/or consumed by the angler. This information could be used in conjunction with the species-specific EPCs to better estimate potential COC intake. The BHHRA evaluated a "mixed fish" diet to account for the presence of multiple fish species in Newark Bay that may be consumed by anglers, which is assumed to comprise equal amounts (20%) of the five species collected as part of the RI/FS (American eel, bluefish, striped bass, summer flounder, and white perch). This application of a "mixed fish" diet in lieu of species-specific ingestion rates may over- or under-estimate consumption.

Crab consumption

USEPA Region 2 evaluated the data collected for the Burger (2002) study in the Newark Bay Complex of New Jersey to estimate crab consumption (USEPA 2012a). The Burger study reported a $50^{th}$ percentile ingestion rate of 3.0 g/day and a $90^{th}$ percentile ingestion rate of 20.9 g/day. As was assumed for fish, crab ingestion rates for the child and adolescent receptors were estimated assuming rates that are one-third and two-thirds of the adult ingestion rates, respectively.

Crab ingestion rates were computed for people who reported only crabbing (i.e., not fishing) and consuming self-caught crabs, resulting in a survey size of 76 respondents.

The following are uncertainties associated with the crab ingestion rate:

- Portion size — USEPA Region 2 assumed that the average edible portion of crab was 45 g/crab for all crabs ingested in the survey. Given natural variability in crab sizes and edible portions, the resulting ingestion rate may be slightly over- or under-estimated. For example, alternative citations of mean edible portions of blue crab include 40.5 g/crab for the Newark Bay Complex and 44.3 g/crab for the Arthur Kill area (Pflugh et al. 2011). Further, in calculating crab ingestion rates, two records were excluded by USEPA because they were considered outliers: one person reported eating 22 crabs per meal 25 times per month, and another person reported eating 48 crabs per meal two times per month. It is clear why the person who reported eating 22 crabs per meal 25 times per month is considered an outlier; that person represents by far the highest number of crabs eaten per month (550, with the next-highest being 182) and the highest daily ingestion rate (135.62 g/day, with the next-highest being 89.8 g/day) among the 76 records. However, it is less clear why the person who reported eating 48 crabs per meal is considered an outlier. Although this person represents the highest number of crabs eaten per meal (48, with the next-highest being 36), they represent only the fifth-highest number of crabs eaten per month (96) and the eighth-highest daily ingestion rate (23.7 g/day) among the 76 records.
- *Recall Bias* — Uncertainty is associated with using single recall survey events (e.g., mail surveys or creel surveys) to estimate long-term consumption rates, because it is difficult for participants to remember their activities over an entire year. Conducting the creel survey during peak season (as in the case of Burger 2002), can result in an overestimation of catch throughout the year and subsequently an overestimation of consumption.

Fraction ingested from contaminated source (fish/crab)

The fraction ingested parameter (FI) represents the fraction of fish and crab consumed by the receptors that is from the NBSA. Although it is possible that anglers/sportsmen catch and consume fish and crab from rivers and other water bodies in the area, the risk assessment conservatively assumes that 100% of the catch is obtained from the NBSA for both the RME and CTE scenarios. USEPA Region 2 assumed that all data reported in Burger (2002) were representative of fishing and crabbing within the Newark Bay Complex. However, it is not clear from Burger (2002) whether anglers reported all fishing or crabbing trips taken in a year (regardless of location) or just Newark Bay trips. If the angler included non-NBSA trips, then an FI assumption of 100% is an overestimate of consumption.

Crab Tissue Type Consumed

The BHHRA assumed that an angler/sportsman would consume both the muscle and hepatopancreas at every crab meal. The basis for this assumption is that some people also eat the hepatopancreas or may use the cooking water after cooking the crab with the hepatopancreas intact. However, as shown in Figure 7-1, the concentrations of PCDD/Fs, DL-PCBs, and non-DL PCBs are on the order of 20 to 40 times higher in the hepatopancreas than in the muscle tissue alone. As a result, the concentrations of these chemicals in muscle and hepatopancreas combined are 7 to 10 times higher than in muscle alone. It is important to note that angler surveys of Newark Bay and coastal New Jersey by the NJDEP indicate that the majority of crabbers remove the hepatopancreas prior to cooking or do not consume it afterward. Further, in regional

surveys conducted in 1995 and 1999, including Newark Bay, Raritan Bay, and coastal New Jersey, 85% to 97% of crabbers reported consuming only crab muscle (May and Burger 1996; NJDEP 2002). Thus, assuming that anglers/sportsmen consume muscle and hepatopancreas tissue at every meal, rather than crab muscle only, as most anglers/sportsmen have been reported to do (May and Burger 1996), likely overestimates the risk to most receptors, because only a small percentage of anglers/sportsmen consumes the hepatopancreas or cooking water. The risks/hazards associated with a crab-muscle-only diet are shown in Figure 7-4. Cancer risk estimates are approximately a factor of 5 to 6 lower than for a combined muscle and hepatopancreas diet; the values for the RME scenario are equal to or just slightly above the NCP risk range whereas the values for the CTE scenario are within the NCP risk range. The noncancer HIs are about a factor of 3 lower, but still higher than the noncancer protection goal of an HI of 1 (USEPA 1991d), even for the CTE scenario. Although not shown in Figure 7-4, the risks/hazards to consumers of the crab hepatopancreas only would be much higher than the risks for consumers of muscle only or combined muscle and hepatopancreas (see Appendix F or G).

Cooking Loss

Loss of hydrophobic COPCs from consumable animal tissue during cooking can have a significant effect on the calculated COPC exposure dose from tissue consumption by humans. The degree of cooking loss can vary with cooking method (e.g., bake, boil, broil, fry, smoke), preparation method (e.g., trimmed/untrimmed, skin-on/skin-off), and animal species. As described in Section 4.3.6.4, the RME scenario for both fish and crab tissue consumption assumes that the consumer ingests the levels of COPCs measured in the raw tissues prior to cooking (i.e., 0% cooking loss), based on uncertainty in cooking methods and the possibility that cooking juices are habitually consumed among some populations. As noted above, however, the assumptions that cooking never results in reduction of COPCs from fish or crab tissue, or that the cooking juices are always consumed, are very conservative. Therefore, fish and crab consumption COPC exposure estimates for the RME scenarios likely resulted in over-estimated cancer risks and noncancer hazards.

The CTE scenario for fish consumption included values for cooking loss ranging from 23% (for DDT) to 57% (for mirex). The CTE scenario for crab consumption assumed 0% cooking loss for all NBSA COPCs. The following table summarizes the results of the available empirical study data on cooking losses of the bioaccumulative organics from fish that constitute the majority of the calculated health risks for the this BHHRA: PCDD/Fs, PCBs, and dieldrin.

| Statistic | PCDD/Fs† | PCBs* | Dieldrin^ |
|---|---|---|---|
| Median | 35% | 28% | 30% |
| Mean | 28% | 26% | 32% |
| Standard Deviation | 31% | 20% | 19% |
| Count of Values | 38 | 107 | 54 |
| Minimum | -59% | -28% | 3% |
| 10th Percentile | -15% | -1% | 8% |
| 25th Percentile | 16% | 15% | 16% |

| Statistic | PCDD/Fs† | PCBs* | Dieldrin^ |
|---|---|---|---|
| 50th Percentile | 35% | 28% | 30% |
| 75th Percentile | 48% | 40% | 43% |
| 90th Percentile | 57% | 49% | 55% |
| Maximum | 100% | 74% | 93% |

[1] Cooking methods included baking/roasting, boiling/poaching, broiling/grilling, frying (deep, pan, wok), microwaving, and smoking. Reported potential COC reduction values from non-cooking preparation methods (e.g., from trimming, dressing, canning) were excluded. See USEPA (2000), AECOM (2012b), and Rawn et al. (2013).

† Cooking loss values reported in AECOM (2012b) and Rawn et al. (2013); three values removed per outlier analysis (Leys et al. 2013).

* Cooking loss values reported in AECOM (2012b) and Rawn et al. (2013); two values removed per outlier analysis (Leys et al. 2013).

^ Cooking loss values reported USEPA (2000); two duplicate values excluded.

The studies summarized by USEPA (2000) and AECOM (2012b), as well as the data reported by Rawn et al. (2013), included a variety of fish species, including striped bass, carp, bass, catfish, perch, trout, flounder, salmon, walleye, and bluefish. Several of these species are relevant to the NBSA. A number of cooking methods were represented, including baking/roasting, boiling/poaching, broiling/grilling, variations on frying (deep, pan, wok), microwaving, and smoking. The degree of cooking loss was variable within and between studies, likely reflecting differences in cooking time, temperature, tissue preparation (skinning and trimming) and fillet geometry, lipid content, initial chemical concentration, analytical method, and extraction efficiency, all factors that are not consistently controlled for across the various studies.

Despite these inconsistencies and the inherent variability in the fish cooking loss data, the database is sufficient to support including cooking loss as a parameter in the CTE quantitative assessment of COPC exposure dose from consumption of fish. Following USEPA's approach of differentiating cooking losses between COPC classes (USEPA 2000), median and mean cooking loss values were computed for the PCDD/Fs, PCBs, and the pesticides recognized as NBSA COPCs for which cooking loss data were available. The cooking loss distributions for PCDD/Fs, PCBs, and dieldrin across cooking methods are illustrated in Figure 7-2, because these COPCs are the largest contributors to NBSA risk estimates of the COPCs with cooking loss data. The following observations are apparent from this analysis:

- For total PCB mixtures, cooking loss ranged from -28% to 74% loss across the 15 studies (Rawn et al. 2013, and the 14 studies relevant studies evaluated in AECOM 2012b). Median losses by cooking method ranged from 11% (boil/poach) to 39% (smoke), with a median of 28% when all PCB data (excluding two outliers) are combined regardless of cooking method.
- For PCDD/Fs, cooking loss ranged from -59% to 100% across five studies (Rawn et al. 2013, and the four relevant studies evaluated in AECOM 2012b). Median losses by cooking method ranged from 16% (bake/roast) to 53% (broil/grill), with a median of 35% when all PCDD/F data (excluding three outliers) are combined regardless of cooking method.
- For dieldrin, cooking loss ranged from 3% to 93% across the relevant studies summarized by USEPA (2000). Median losses by cooking method ranged from 16% (boil/poach) to 58% (pan fry), with a median of 30% when all dieldrin data are combined regardless of cooking method.

Finally, the literature features very little empirical cooking loss data for the NBSA COPCs and crab consumption. The default assumption that no cooking loss occurs from crab tissue for the organochlorine COPCs likely overestimates crab consumption risks for these compounds: the limited data demonstrate an approximately 20% reduction of PCBs in boiled/steamed blue crab, and the overlap in general physicochemical properties suggests that the distribution of other organochlorine COPCs within crab tissues in response to cooking temperatures is likely similar to that of PCBs.

#### *7.2.2.3 Consumption of Other Fish/Crab Diets*

Estimated cancer risks and noncancer hazards are presented in the BHHRA for an angler/sportsman assumed to consume a diet of self-caught fish (composed of equal portions of American eel, blue fish, striped bass, summer flounder, and white fish) or self-caught crab (muscle and hepatopancreas combined). However, it is possible that some anglers/sportsmen will have a preference for a particular fish species and will limit consumption to that single species. Cancer risks and noncancer hazards for the adult angler based on a mixed fish diet and single-species diets for the five fish species that make up the mixed fish diet are presented in Figure 7-3 for both the RME and CTE scenarios. As shown in the figure, the RME cancer risks for all diets are above $10^{-4}$, ranging from approximately $3\times10^{-4}$ (bluefish) to approximately $1\times10^{-3}$ (American eel striped bass, and white perch), as compared to approximately $8\times10^{-4}$ for a mixed fish diet. For the CTE scenario, all cancer risks are within the NCP risk range. The RME noncancer HIs for all diets are above the noncancer protection goal of an HI of 1 (USEPA 1991d), ranging from approximately 20 for blue fish to approximately 60 for American eel and striped bass, as compared to approximately 40 for a mixed fish diet. The noncancer HIs for the CTE scenario are also above 1 for all diets, but about a factor of 10 lower than those for the RME scenario.

It is also possible that some anglers/sportsmen will consume both fish and crab. According to Burger (2002), most Newark Bay anglers consumed only fish or only crab, with only approximately 12% reporting that they consumed both. Further, anglers/sportsmen who fished and crabbed reported eating fish and crab more frequently than those who did not (average of 6 times per month vs. 4 times or fewer). These data suggest that those anglers/sportsmen who consume both fish and crab from NBSA may be at a higher risk than anglers/sportsmen who consume only fish or only crab.

### 7.2.3 Estimation of Exposure-Point Concentrations

The EPCs are based on data assumed to be representative of site conditions. For example, the sediment samples used in the BHHRA were collected in areas that were reasonably accessible by workers or recreational users around the perimeter of the NBSA. Surface water samples were collected over multiple years and flow conditions. Samples of five fish species and two crab tissue types were collected to reflect the local fish community and angler preferences. Despite these efforts, there is uncertainty in the EPCs used to represent current (and future) conditions because of the large size of the NBSA and the temporal and spatial variability in the ecosystem.

USEPA (1989) guidance states that the 95% upper confidence limit (UCL) on the arithmetic mean concentration should be used as the EPC, because this statistic represents a reasonable upper bound on

the arithmetic average concentration that is contacted over the exposure period. The 95% UCL was used as the EPC for both the RME and CTE scenarios, unless it was greater than the maximum concentration; in those few cases, the maximum concentration was used as the EPC. Accordingly, it is unlikely that the EPCs used in the BHHRA underestimate actual exposure concentrations over an extended period of time.

#### *7.2.3.1 Uncertainty in Sediment EPCs*

The accessible surface sediment data used to estimate EPCs are from 39 locations across the NBSA; field duplicate samples were collected at two locations. These data were collected as part of two sampling programs: Crab and Clam Sampling Program (16 locations) and SQT and Porewater Sampling Program (23 locations). While this is not a large number of samples given the size of the NBSA, the analytical results from the two sampling programs were reasonably similar, which adds confidence to the overall representativeness of the data. For example, for PCDD/Fs and PCBs, the concentrations between the two sampling programs are generally within a factor of two.

#### *7.2.3.2 Uncertainty in Surface Water EPCs*

The surface water data used to estimate EPCs are from six locations across the NBSA. Samples were collected over seven sampling rounds under normal and high-flow conditions. A total of 131 samples were collected in the upper 3 feet of the surface water column, which is assumed to be the depth to which recreational users would be exposed. Additional data were collected within the bottom 3 feet of the water column. Although not used to calculate EPCs, the average concentrations for near-surface samples are generally within 50% of the average concentrations for all surface samples. This consistency between the near-surface and deeper surface water concentrations adds confidence to the overall representativeness of the surface water EPCs.

#### *7.2.3.3 Uncertainty in Tissue EPCs*

The fish tissue data used in the BHHRA are from 95 fish filet samples across five species collected during 2014, 2015, and 2017. For crab tissue, the data include 37 samples each of Blue crab muscle and hepatopancreas collected in 2014. The EPC for a mixed fish diet was calculated by dividing the EPC for each species by 5, and then taking the sum. This assumes that a mixed fish diet comprises exactly equal portions of all five species, without regard to prevalence or the length of time that each species is resident in Newark Bay. The uncertainty in this assumption is addressed by estimating risks for single-species diets for comparison purposes (see Section 7.2.2.3). For crab, the BHHRA assumed that anglers/sportsman consumed both the crab muscle and hepatopancreas. Because these tissue types were analyzed separately, the concentration in crab muscle and hepatopancreas combined had to be estimated based on the relative weights of the two tissues, as measured in 34 of the 37 crabs collected. From these data, it was estimated that the crab muscle constitutes 76%, and the crab hepatopancreas 24%, of the combined tissue. While there is some uncertainty associated with this calculation, as opposed to analyzing the combined tissue, the amount of uncertainty is expected to be small.

#### *7.2.3.4 Assumption of No Degradation*

For purposes of the BHHRA, the EPCs calculated based on current site conditions are assumed to remain the same for the entire exposure duration, which is up to 26 years for the combined adult/child receptor. This is an inherently conservative assumption, because chemicals in the environment are subject to natural processes, including biodegradation and attenuation. The extent of any degradation will depend on various chemical-specific and environmental factors; however, by assuming no degradation at all over such an extended period of time, the estimated cancer risks and noncancer hazards are likely to be overestimated, at least to some degree.

#### *7.2.3.5 Methods and Assumptions Used to Model Media Concentrations*

The only exposure pathway evaluated that relied on a model was inhalation of vapors from sediment or surface water (see Appendix C-1 and C-2, respectively). In the absence of empirical data, screening-level models were used to estimate potential volatilization of VOCs and SVOCs into ambient air and subsequent dispersion to a downwind (most highly exposed) receptor. The results of these screening-level assessments indicate that inhalation of ambient air adjacent to the NBSA is not of concern, and it is unlikely that these risks have been underestimated.

### 7.2.4 Estimation of Exposure Dose

Dermal absorption fractions and oral bioavailability factors, where available, were used to account for differences between exposure conditions for humans vs. laboratory animals. The uncertainty in these assumptions is discussed below.

#### *7.2.4.1 Default Dermal Absorption Fractions*

As discussed in Section 4.3.10.1, default dermal absorption fractions (DAFs) were compiled from RAGS Part E (USEPA 2004b), because site-specific information was not available. These values were derived by USEPA to be conservative for most sites, but may be overly conservative, especially for lipophilic compounds such as PCDD/Fs and PCBs, at some sites with high organic carbon content. For example, USEPA (2004b) provides two DAFs for PDDD/Fs, the default value being 0.03, but an alternative value of 0.001 (30-fold lower) when the fraction of organic carbon is greater than 10%. Nevertheless, use of the default DAFs did not result in estimated cancer risks or noncancer hazards that exceeded the NCP risk range or noncancer protection goal of 1 (USEPA 1991d).

#### *7.2.4.2 Oral Bioavailability*

A value of 1 (100%) was assumed for the oral relative bioavailability (RBA) factor for all COPCs in sediment except arsenic, for which a value of 0.6 (60%) was assumed, consistent with USEPA approaches (1989, 2019a). The extent to which the RBA for chemicals other than arsenic has been overestimated is unknown; however, USEPA has discussed methods for estimating the RBA of PCDD/Fs in soil, and the available studies suggest that the evidence supports a value less than 100% (USEPA 2010c). Regardless, use of

100% RBA for all COPCs other than arsenic did not result in estimated cancer risks or noncancer hazards that exceeded the NCP risk range or noncancer protection goal of 1 (USEPA 1991d). As noted in Section 4.3.12, a default RBA of 0.6 (60%) was assumed for arsenic in sediment. This value was based on empirical measurements from over 100 studies, in which less than 5% of the studies measured RBAs greater than 60% (USEPA 2012c). As noted by USEPA, there is uncertainty associated with using this national default value in lieu of site-specific RBA estimates, and risks may be over- or underestimated. There may be additional uncertainty associated with using this value for estimating exposure to arsenic in sediment because only two of the studies upon tested sediment samples, although the RBA measured in these studies was 38 +/-2% and 52 +/-2% (USEPA 2012g). The estimated cancer risks associated with exposure to arsenic in sediment were at the low end of the NCP risk range; therefore, the uncertainty in this value is not expected to affect the overall conclusions of the BHHRA.

## 7.3 Toxicity Assessment

The purpose of the toxicity assessment is to determine the nature of adverse health effects that may occur with exposure to a certain chemical, and to identify the relationship between the dose of a chemical and the possibility and extent of a potential adverse effect (USEPA 1989). Adverse effects are divided into two categories—cancer and noncancer—where cancer effects are generally thought to occur by a linear, no-threshold mode of action (zero risk at zero dose), whereas noncancer effects are generally thought to occur by a nonlinear threshold mode of action. USEPA has developed a series of guidelines for deriving toxicity values for these two classes of compounds (e.g., USEPA 2002b, 2005b, 2012f). These methods have inherently many of the same uncertainties, given the limited understanding of the toxicity to humans exposed to substances at the low concentrations generally encountered in the environment. Accordingly, USEPA relies on conservative methods and assumptions to extrapolate from high-dose animal studies to predict the possible response in humans at exposure levels far below those administered to animals. Even in cases where human exposure data are available, these data are often from high-exposure settings, such as the workplace, that are not representative of much lower exposure levels found in the environment. Overall, uncertainty in the toxicity values used to estimate risk is often the largest source of uncertainty in the entire risk assessment. Depending on the nature of the data used to develop the toxicity values, there are some under or overestimates of risks and hazards for the individual chemicals.

### 7.3.1 Evaluation of Noncarcinogenic Dose-Response

Often, toxicity factors are based on animal studies, because human health effects data are not available for many chemicals. Seventy-four of the 82 COPCs quantitatively evaluated (this includes the individual PCDD/PCDF and DL-PCB congeners) have oral RfDs. Of these 74, 34 are based on animal data, and 40 are based on human toxicological information. (Of the 40 COPCs that are based on human studies, a large number [28] are individual PCDD/PCDF and DL-PCB congeners.) USEPA's risk assessment guidelines include the assumption that animal data are relevant for human exposures (USEPA 1989, 1991e, 2002b). As indicated above, extrapolation of animal toxicity information to humans adds uncertainty to the risk characterization step, and when human data are available, uncertainty is decreased. Further, uncertainty is increased when the mechanism and fate for a chemical are unknown; particularly if the mechanism and fate are unknown in humans.

UFs are generally used to estimate human responses from animal toxicity data. UFs are intended to be health protective, with toxicity factors intentionally overestimating toxicity in humans. RfDs are based on toxicity data representing the most sensitive species and the lowest dose causing a mild effect, when information is available. UFs are applied to this lowest dose level. UFs typically address the length of study, and interspecies and intraspecies variability, LOAEL-to-NOAEL extrapolation, and database uncertainty. Individual UFs range from 1 to 10; USEPA (2002b) recommends that the combined UF for a chemical not exceed 3,000. In this BHHRA, combined UFs for the COPCs are within this range (1 for manganese, up to 3,000 for cobalt, 2,4'-DDE [based on 4,4'-DDE as a surrogate], 4,4'-DDE, naphthalene, and thallium). If UFs are artificially high (e.g., human toxicity is more similar to the species used to derive the toxicity factor, the dose needed to induce chronic toxicity is similar to the subchronic dose), hazard may be overestimated. Alternatively, if UFs are lower than necessary (e.g., the most sensitive species was not selected for the toxicity studies), hazard may be underestimated.

Adverse effects occurring in animals may not materialize in humans, due to different fate and metabolic processes between species. This could lead to an overestimation of toxicity in humans and a resulting RfD that is lower than intended. It is acknowledged that, even with these layers of protectiveness, there is the chance that an animal study will not show a toxic effect that could occur in humans. This phenomenon could potentially lead to underestimating the chemical's toxicity in humans, and a resulting RfD that is higher than would be considered adequately protective.

Of the eight COPCs that do not have oral RfDs, six are the carcinogenic PAHs other than BaP. Lead does not have an RfD but is evaluated in risk assessments using blood lead modeling (see Appendix E). Titanium uses titanium tetrachloride as a surrogate and lacks an oral RfD. Chemicals lacking RfDs lead to a potential underestimate of total hazards.

### 7.3.2 Evaluation of Carcinogenic Dose-Response

There is also uncertainty in estimating dose-response relationships for potential carcinogens, perhaps more so than for the noncarcinogens discussed above. The primary sources of this uncertainty include (1) selection of the underlying study, (2) conversion from animal to human dose, when necessary, and (3) mathematical model used for high-dose to low-dose extrapolation. Of the 82 chemicals/chemical groups identified as COPCs, 58 are classified as carcinogenic or potentially carcinogenic via the oral route.

#### *7.3.2.1 Study Selection*

In general, study selection involves a process of evaluating the available toxicity data to identify a data set that provides sufficient dose-response information to support derivation of a defensible CSF. When available, human epidemiology are preferred; however, in most cases, adequate human data are lacking, and it is necessary to rely on laboratory animal data instead. Ideally, the animal study is in a species that reasonably resembles humans biologically and where the administration route is the same as or similar to the expected route of human exposure. The study selection criteria, in combination, are intended to be health-protective, such that the resulting dose-response assessment is more likely to overstate, rather than understate, the potential cancer risk. If a species substantially more sensitive than humans to carcinogenic

effects of a particular chemical was used for the primary study, risk may be overestimated. Alternatively, risk could be underestimated if the most sensitive species was not used for the study.

Of the chemicals identified as potential COCs, arsenic is the only one for which the oral CSF is based on human epidemiological studies, and arsenic contributes, at most, only a few percent to the total cumulative cancer risk. Except for benzo(a)pyrene, which was identified as a potential COC but contributes less than 1% to the total cumulative risk, the remaining potential COCs that are carcinogens are classified as "B2," which is defined as sufficient evidence of carcinogenicity in animals with inadequate or a lack of evidence in humans under the 1986 cancer classification scheme (USEPA 1986b). Benzo(a)pyrene is classified as "carcinogenic to humans" under the current classification scheme (USEPA 2005b), but as noted, is only a very small contributor to the total cumulative risk. If there are potential COCs which are in fact carcinogenic but lack a CSF, total risks would be underestimated.

As identified in Section 5.1, a Tier-3 oral CSF was used for 2,3,7,8-TCDD, because there is no currently recommended value on IRIS, nor is there a PPRTV. This value is from USEPA's HEAST (1997a), based on a dietary study in rats, and is used as the index for the remaining PCDD/Fs and DL-PCBs, which in total, constitute the majority of the total cumulative cancer risk. The uncertainty in this value is discussed in Section 7.3.6.

The remaining potential COC that is a primary contributor to the total cumulative cancer risk is non-DL PCBs. This group of compounds has been shown to cause cancer in animals, and the CSFs for PCBs provided in IRIS are also based on a dietary study in rats (USEPA 1996b, 2019b). In addition, while IRIS considers the human carcinogenicity data for PCBs to be "inadequate, but suggestive," the weight-of-evidence classification as of the last IRIS review in 1996 is "B2" (sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans) (USEPA 2019b). Since that time, the International Agency for Research on Cancer (IARC) has concluded that available evidence in humans is sufficient to classify PCBs as Group 1 (carcinogenic in humans (IARC 2016).

#### *7.3.2.2 Interspecies Dose Conversion*

In USEPA's calculation of human equivalent doses from animal data, it is assumed that animals and humans have the same sensitivity to a chemical's toxic effects—provided the mechanism of toxicity is identical and the same amount of substance per body surface area is absorbed by both animals and humans. This evaluation of the particular animal species relative to humans is extremely useful, in that it yields information regarding the shape of the dose-response curve for doses at which tumors occur, includes assessment of biomarkers of health effects, identifies levels where carcinogenic impacts are occurring, and facilitates interspecies extrapolations when data are available from both human and animal cells (USEPA 2005b). Although determination of upper bounds does not rely on susceptibility information, using upper bounds is typically regarded as a conservative, protective approach for accounting for risk to sensitive persons. However, USEPA (2005b) indicates that dose conversions between species typically yield CSFs that do not represent a risk for a highly sensitive subpopulation or individual, but are usually an upper bound on risk for a randomly selected person, or the average risk in a population.

Additional assumptions (and added uncertainties) are involved in assessment of risks based on one exposure route (e.g., ingestion) when the original study used a different exposure route (e.g., inhalation). Scaling factors are employed to handle disparities between animals and humans regarding breathing rates, body size, life span, and other physiological differences. Although updates to the older CSFs will be made when the USEPA updates toxicity values using the IRIS procedures, USEPA has altered its default recommendation for scaling animal data to humans; scaling is now recommended as a per-body-weight basis instead of a per-surface-area basis (USEPA 1992b, 2005b). Note that USEPA's 1996 cancer assessment for PCBs (USEPA 1996b) includes an extrapolation of body weight to the three-quarters power.

#### *7.3.2.3 High-Dose to Low-Dose Extrapolation*

The concentrations tested in animal toxicity studies are generally much higher than what humans are exposed to in the environment. Accordingly, estimating health effects at these low dose levels requires the use of models to extrapolate effects observed in high-dose animal studies, which introduces uncertainty in the dose-response assessment. There are many different forms of these mathematical models, depending on the type of data being analyzed, but they are designed and applied in such a way as to more likely overstate, rather than understate, the potential cancer risk. For example, many of the CSFs provided in the IRIS database are based on the 95% UCL of the slope predicted by the linearized multistage (LMS) model, which assumes that some risk exists at any level of exposure. This value represents the plausible upper limit to the risk, consistent with some proposed carcinogenic mechanisms; however, as acknowledged by USEPA, the true risk is unknown and may be zero (USEPA 1989, 2005b). Use of these upper-bound cancer potency estimates is expected to result in conservative (i.e., health-protective) estimates of potential cancer risk.

### 7.3.3 Uncertainty in TEF Approach

USEPA recommends using the TEF method to assess health risks posed by mixtures of DLCs (USEPA 2010a). This method provides a means of estimating the combined toxicity of DLCs by scaling each DLC's dose according to its relative potency, and summing across all DLCs. The scaling factors recommended by USEPA are the 2005 WHO consensus TEFs (USEPA 2010a; van den Berg et al. 2006). These TEFs represent single estimates of relative potency; however, a wide range of relative potencies exists in the literature for each of the DLCs (USEPA 2010a; Haws et al. 2006). Therefore, for risk assessments, USEPA recommends conducting a sensitivity analysis to illustrate the impact of the TEFs.

USEPA recommends the following methods for evaluating lower- and upper-bound TEFs (i.e., $TEF_{iU}$ and $TEF_{iL}$) (USEPA 2010a):

1. $TEF_{iL}$ and $TEF_{iU}$ can be defined by dividing and multiplying the WHO 2005 TEFs by half a log (i.e., 3.16), respectively.
2. $TEF_{iL}$ and $TEF_{iU}$ can be based on statistical summaries of ReP data. Suggested statistical summaries include the minimum/maximum, $10^{th}$/$90^{th}$ percentiles, or interquartile ranges.

In this sensitivity analysis, the 10th and 90th percentiles of *in vitro* and *in vivo* ReP data, shown in Table 4 of USEPA (2010a), were selected to represent $TEF_{iL}$ and $TEF_{iU}$, respectively. For the coeluting congeners PCB-156 and PCB-157, the maximum of the 10th and 90th percentiles for either of these congeners was conservatively selected to represent $TEF_{iL}$ and $TEF_{iU}$ (i.e., $TEF_{PCB\text{-}156/157,L}$= 0.0001 and $TEF_{PCB\text{-}156/157,U}$= 0.2). The lower- and upper-bound TEFs used in the sensitivity analysis, as well as the default WHO 2005 TEFs, are shown in the table below. Additionally, ratios comparing the $TEF_{iL}$ and $TEF_{iU}$ to the WHO 2005 TEFs can be seen.

**Toxic Equivalence Factors (TEFs) for Dioxin-Like Compounds**

| Chemical of Potential Concern | $TEF_{iL}$ | $TEF_{WHO}$ | $TEF_{iU}$ | Ratio of TEF ($TEF_{iL}$/ $TEF_{WHO}$) | Ratio of TEF ($TEF_{iU}$/ $TEF_{WHO}$) |
|---|---|---|---|---|---|
| 2,3,7,8-TCDD | 1 | 1 | 1 | 1.0 | 1.0 |
| 1,2,3,7,8-PeCDD | 0.1 | 1 | 0.8 | 0.1 | 0.8 |
| 1,2,3,4,7,8-HxCDD | 0.04 | 0.1 | 0.4 | 0.4 | 4.0 |
| 1,2,3,6,7,8-HxCDD | 0.03 | 0.1 | 0.1 | 0.3 | 1.0 |
| 1,2,3,7,8,9-HxCDD | 0.02 | 0.1 | 0.07 | 0.2 | 0.7 |
| 1,2,3,4,6,7,8-HpCDD | 0.004 | 0.01 | 0.04 | 0.4 | 4.0 |
| OCDD | 0.0003 | 0.0003 | 0.003 | 1.0 | 10.0 |
| 2,3,7,8-TCDF | 0.01 | 0.1 | 0.3 | 0.1 | 3.0 |
| 1,2,3,7,8-PeCDF | 0.01 | 0.03 | 0.1 | 0.3 | 3.3 |
| 2,3,4,7,8-PeCDF | 0.05 | 0.3 | 1 | 0.2 | 3.3 |
| 1,2,3,4,7,8-HxCDF | 0.04 | 0.1 | 0.5 | 0.4 | 5.0 |
| 1,2,3,6,7,8-HxCDF | 0.01 | 0.1 | 0.1 | 0.1 | 1.0 |
| 1,2,3,7,8,9-HxCDF | 0.1 | 0.1 | 0.2 | 1.0 | 2.0 |
| 2,3,4,6,7,8-HxCDF | 0.01 | 0.1 | 0.3 | 0.1 | 3.0 |
| 1,2,3,4,6,7,8-HpCDF | 0.05 | 0.01 | 0.3 | 5.0 | 30.0 |
| 1,2,3,4,7,8,9-HpCDF | 0.02 | 0.01 | 0.04 | 2.0 | 4.0 |
| OCDF | 0.00003 | 0.0003 | 0.002 | 0.1 | 6.7 |
| PCB-77 | 0.00002 | 0.0001 | 0.1 | 0.2 | 1000.0 |
| PCB-81 | 0.0006 | 0.0003 | 0.02 | 2.0 | 66.7 |
| PCB-105 | 0.000005 | 0.00003 | 0.002 | 0.2 | 66.7 |
| PCB-114 | 0.0002 | 0.00003 | 0.002 | 6.7 | 66.7 |
| PCB-118 | 0.000002 | 0.00003 | 0.002 | 0.1 | 66.7 |
| PCB-123 | 0.00001 | 0.00003 | 0.0004 | 0.3 | 13.3 |
| PCB-126 | 0.01 | 0.1 | 0.4 | 0.1 | 4.0 |
| PCB-156/157 | 0.0001 | 0.00003 | 0.2 | 3.3 | 6666.7 |

| Toxic Equivalence Factors (TEFs) for Dioxin-Like Compounds | | | | | |
|---|---|---|---|---|---|
| **Chemical of Potential Concern** | **$TEF_{iL}$** | **$TEF_{WHO}$** | **$TEF_{iU}$** | **Ratio of TEF ($TEF_{iL}$/ $TEF_{WHO}$)** | **Ratio of TEF ($TEF_{iU}$/ $TEF_{WHO}$)** |
| PCB-167 | 0.000005 | 0.00003 | 0.0004 | 0.2 | 13.3 |
| PCB-169 | 0.0007 | 0.03 | 0.5 | 0.0 | 16.7 |
| PCB-189 | 0.000005 | 0.00003 | 0.0001 | 0.2 | 3.3 |

The lower-bound TEF, $TEF_{iL}$, differs by a factor of 10 or more from the default WHO 2005 TEF for the following compounds:

- Decrease: 1,2,3,7,8-PeCDD, 2,3,7,8-TCDF, 1,2,3,6,7,8-HxCDF, OCDF, PCB-118, PCB-126, PCB-169
- Increase: none of the compounds increased by more than a factor of 10.

The greatest difference in TEF was noted for PCB-169, for which the default TEF is 0.03 and the $TEF_{iL}$ is 0.0007.

The upper-bound TEF, $TEF_{iU}$, differs by a factor of 10 or more relative to the default WHO TEF for the following compounds:

- Decrease: none of the compounds decreased by more than a factor of 10
- Increase: OCDD, 1,2,3,4,6,7,8-HpCDF, PCB-77, PCB-81, PCB-105, PCB-114, PCB-118, PCB-123, PCB-156/157, PCB-167, PCB-169.

The greatest differences in TEF were noted for PCB-77 and PCB-156/157, which changed by factors of 1000 and 6667, respectively. These lower- and upper-bound TEFs were used to calculate excess cancer risk for angler consumption of fish and crab.

TEF sensitivity analysis — Angler/sportsman consumption of a mixed fish diet

RME risk from the angler/sportsman's consumption of a mixed fish diet is shown in the table below. Risks are presented for each of the individual DLCs and non-DL PCBs, as well as aggregates by chemical type (i.e., Total DLCs, Total PCDD/Fs, Total DL-PCBs, Total PCBs [dioxin-like and non-dioxin-like], and overall Total risk [from all potential COCs, not just dioxin/furans and PCBs]).

The overall risk for this pathway using the default WHO 2005 TEF values for DLCs is $8\times10^{-4}$. When using lower-bound estimates of TEFs for DLCs, the overall risk reduces to $5\times10^{-4}$. Employing upper-bound estimates of TEFs for DLCs increases the overall risk dramatically, to $2\times10^{-2}$. The largest difference in risk was noted for PCB-156/157, which went from $2.3\times10^{-6}$ in the default TEF evaluation to $1.5\times10^{-2}$ when using $TEF_{iU}$. Additionally, large increases in risk were noted for PCB-77 and PCB-118 for the $TEF_{iU}$ evaluation.

Risks for 2,3,7,8-TCDD are constant across the $TEF_{iL}$, $TEF_{WHO}$ (default), and $TEF_{iU}$ evaluations (i.e., $2.2\times10^{-4}$), because the TEF is equal to 1 in all scenarios. However, the contribution to risk changes for 2,3,7,8-TCDD drastically, ranging from 44% of the total risk when the lower-bound TEFs are used for all DLCs, to 1% of the total risk when the upper-bound TEFs are used for all DLCs. Conversely, the percent contribution to overall risk for Total PCBs (DL-PCBs and Non-DL PCBs) increases from 37% when using lower-bound TEFs to 98% when upper-bound TEFs are used.

**Angler/Sportsman (Adult + Child) RME Risk from Consumption of Mixed Fish (all species) Based on Various TEFs ($TEF_{iL}$, $TEF_{WHO}$, and $TEF_{iU}$)**

| Receptor | Expo Pathway | Chemical of Potential Concern | $TEF_{iL}$ | | $TEF_{WHO}$ | | $TEF_{iU}$ | |
|---|---|---|---|---|---|---|---|---|
| | | | Risk | Contribution to Total Risk (%) | Risk | Contribution to Total Risk (%) | Risk | Contribution to Total Risk (%) |
| Angler Adult/Child | Mixed Fish/All Species | **Dioxin-Like Compounds** | | | | | | |
| | | 2,3,7,8-TCDD | 2.2E-04 | 44.3% | 2.2E-04 | 28.4% | 2.2E-04 | 1.1% |
| | | 1,2,3,7,8-PeCDD | 1.2E-06 | 0.2% | 1.2E-05 | 1.5% | 9.6E-06 | 0.0% |
| | | 1,2,3,4,7,8-HxCDD | 2.2E-07 | 0.0% | 5.6E-07 | 0.1% | 2.2E-06 | 0.0% |
| | | 1,2,3,6,7,8-HxCDD | 5.2E-07 | 0.1% | 1.7E-06 | 0.2% | 1.7E-06 | 0.0% |
| | | 1,2,3,7,8,9-HxCDD | 8.2E-08 | 0.0% | 4.1E-07 | 0.1% | 2.9E-07 | 0.0% |
| | | 1,2,3,4,6,7,8-HpCDD | 7.7E-08 | 0.0% | 1.9E-07 | 0.0% | 7.7E-07 | 0.0% |
| | | OCDD | 3.4E-08 | 0.0% | 3.4E-08 | 0.0% | 3.4E-07 | 0.0% |
| | | 2,3,7,8-TCDF | 4.6E-07 | 0.1% | 4.6E-06 | 0.6% | 1.4E-05 | 0.1% |
| | | 1,2,3,7,8-PeCDF | 4.2E-07 | 0.1% | 1.2E-06 | 0.2% | 4.2E-06 | 0.0% |
| | | 2,3,4,7,8-PeCDF | 3.3E-06 | 0.7% | 2.0E-05 | 2.5% | 6.6E-05 | 0.3% |
| | | 1,2,3,4,7,8-HxCDF | 1.2E-06 | 0.2% | 3.0E-06 | 0.4% | 1.5E-05 | 0.1% |
| | | 1,2,3,6,7,8-HxCDF | 2.7E-07 | 0.1% | 2.7E-06 | 0.3% | 2.7E-06 | 0.0% |
| | | 1,2,3,7,8,9-HxCDF | 3.1E-07 | 0.1% | 3.1E-07 | 0.0% | 6.2E-07 | 0.0% |
| | | 2,3,4,6,7,8-HxCDF | 4.6E-08 | 0.0% | 4.6E-07 | 0.1% | 1.4E-06 | 0.0% |
| | | 1,2,3,4,6,7,8-HpCDF | 3.2E-06 | 0.6% | 6.3E-07 | 0.1% | 1.9E-05 | 0.1% |
| | | 1,2,3,4,7,8,9-HpCDF | 5.9E-08 | 0.0% | 2.9E-08 | 0.0% | 1.2E-07 | 0.0% |
| | | OCDF | 1.9E-10 | 0.0% | 1.9E-09 | 0.0% | 1.2E-08 | 0.0% |
| | | PCB-77 | 2.7E-07 | 0.1% | 1.3E-06 | 0.2% | 1.3E-03 | 6.5% |
| | | PCB-81 | 3.1E-07 | 0.1% | 1.6E-07 | 0.0% | 1.0E-05 | 0.1% |
| | | PCB-105 | 1.0E-06 | 0.2% | 6.2E-06 | 0.8% | 4.1E-04 | 2.0% |
| | | PCB-114 | 3.2E-06 | 0.6% | 4.8E-07 | 0.1% | 3.2E-05 | 0.2% |
| | | PCB-118 | 1.7E-06 | 0.3% | 2.5E-05 | 3.2% | 1.7E-03 | 8.2% |
| | | PCB-123 | 1.5E-07 | 0.0% | 4.6E-07 | 0.1% | 6.1E-06 | 0.0% |
| | | PCB-126 | 2.5E-05 | 4.8% | 2.5E-04 | 31.1% | 9.8E-04 | 4.8% |

| Angler/Sportsman (Adult + Child) RME Risk from Consumption of Mixed Fish (all species) Based on Various TEFs ($TEF_{iL}$, $TEF_{WHO}$, and $TEF_{iU}$) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Receptor** | **Expo Pathway** | **Chemical of Potential Concern** | **$TEF_{iL}$** | | **$TEF_{WHO}$** | | **$TEF_{iU}$** | |
| | | | **Risk** | **Contribution to Total Risk (%)** | **Risk** | **Contribution to Total Risk (%)** | **Risk** | **Contribution to Total Risk (%)** |
| | | PCB-156/157 | 7.7E-06 | 1.5% | 2.3E-06 | 0.3% | 1.5E-02 | 75.0% |
| | | PCB-167 | 1.7E-07 | 0.0% | 1.0E-06 | 0.1% | 1.3E-05 | 0.1% |
| | | PCB-169 | 7.2E-08 | 0.0% | 3.1E-06 | 0.4% | 5.2E-05 | 0.3% |
| | | PCB-189 | 3.1E-08 | 0.0% | 1.8E-07 | 0.0% | 6.1E-07 | 0.0% |
| | | **Non-DL PCBs** | | | | | | |
| | | Total Non-DL PCBs | 1.5E-04 | 29.3% | 1.5E-04 | 18.8% | 1.5E-04 | 0.7% |
| | | **Total DLCs** | 3E-04 | 54.3% | 6E-04 | 70.7% | 2E-02 | 98.9% |
| | | **Total PCDD/Fs** | 2E-04 | 46.6% | 3E-04 | 34.5% | 4E-04 | 1.8% |
| | | **Total DL-PCBs** | 4E-05 | 7.7% | 3E-04 | 36.2% | 2E-02 | 97.1% |
| | | **Total PCBs (DL & NDL)** | 2E-04 | 37.0% | 4E-04 | 55.0% | 2E-02 | 97.8% |
| | | **TOTAL - All potential COCs** | 5E-04 | 100.0% | 8E-04 | 100.0% | 2E-02 | 100.0% |

TEF sensitivity analysis — Angler/sportsman consumption of crab muscle and hepatopancreas

RME risk from the angler/sportsman's consumption of crab muscle and hepatopancreas is shown in the table below. Risks are presented for each of the individual DLCs and non-DL PCBs, as well as aggregates by chemical type (i.e., Total DLCs, Total PCDD/Fs, Total DL-PCBs, Total PCBs [dioxin-like and non-dioxin-like], and overall Total risk [from all potential COCs, not just dioxin/furans and PCBs]).

The results of RME risk for the crab muscle and hepatopancreas consumption are similar to the RME risk for a mixed fish diet. Overall risk for this pathway using default WHO 2005 TEF values for DLCs is $8\times10^{-4}$. When using lower-bound estimates of TEFs for DLCs, the overall risk reduces to $6\times10^{-4}$. Employing upper-bound estimates of TEFs for DLCs increases the overall risk dramatically, to $2\times10^{-2}$. The largest difference in risk was noted for PCB-156/157, which went from $1.8\times10^{-6}$ in the default TEF evaluation to $1.2\times10^{-2}$ when using $TEF_{iU}$.

Risks for 2,3,7,8-TCDD are constant across the $TEF_{iL}$, $TEF_{WHO}$ (default), and $TEF_{iU}$ evaluations (i.e., $4.3\times10^{-4}$), because the TEF is equal to 1 in all scenarios. However, the contribution to risk changes drastically for 2,3,7,8-TCDD, ranging from 70% of the total risk when the lower-bound TEFs are used for all DLCs, to 2% of the total risk when the upper-bound TEFs are used for all DLCs. Contribution to risk for Total PCDD/Fs decreases from 73% to 3% when comparing lower- and upper-bound TEFs, whereas DL-PCBs contribution to risk increases from 5% to 96%.

| Angler (Adult + Child) RME Risk from Consumption of Crab (hepatopancreas + muscle) Based on Various TEFs ($TEF_{iL}$, $TEF_{WHO}$, and $TEF_{iU}$) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Receptor** | **Expo Pathway** | **Chemical of Potential Concern** | **$TEF_{iL}$** | | **$TEF_{WHO}$** | | **$TEF_{iU}$** | |
| | | | **Risk** | **Contribution to Total Risk (%)** | **Risk** | **Contribution to Total Risk (%)** | **Risk** | **Contribution to Total Risk (%)** |
| **Angler Adult/Child** | **Crab (H+M)** | **Dioxin-Like Compounds** | | | | | | |
| | | 2,3,7,8-TCDD | 4.3E-04 | 70.1% | 4.3E-04 | 51.6% | 4.3E-04 | 2.3% |
| | | 1,2,3,7,8-PeCDD | 1.1E-06 | 0.2% | 1.1E-05 | 1.4% | 9.1E-06 | 0.0% |
| | | 1,2,3,4,7,8-HxCDD | 1.3E-07 | 0.0% | 3.3E-07 | 0.0% | 1.3E-06 | 0.0% |
| | | 1,2,3,6,7,8-HxCDD | 2.9E-07 | 0.0% | 9.7E-07 | 0.1% | 9.7E-07 | 0.0% |
| | | 1,2,3,7,8,9-HxCDD | 6.9E-08 | 0.0% | 3.5E-07 | 0.0% | 2.4E-07 | 0.0% |
| | | 1,2,3,4,6,7,8-HpCDD | 4.5E-08 | 0.0% | 1.1E-07 | 0.0% | 4.5E-07 | 0.0% |
| | | OCDD | 9.9E-09 | 0.0% | 9.9E-09 | 0.0% | 9.9E-08 | 0.0% |
| | | 2,3,7,8-TCDF | 1.6E-06 | 0.3% | 1.6E-05 | 1.9% | 4.8E-05 | 0.3% |
| | | 1,2,3,7,8-PeCDF | 3.5E-07 | 0.1% | 1.0E-06 | 0.1% | 3.5E-06 | 0.0% |
| | | 2,3,4,7,8-PeCDF | 4.0E-06 | 0.6% | 2.4E-05 | 2.9% | 8.0E-05 | 0.4% |
| | | 1,2,3,4,7,8-HxCDF | 3.1E-06 | 0.5% | 7.7E-06 | 0.9% | 3.9E-05 | 0.2% |
| | | 1,2,3,6,7,8-HxCDF | 2.3E-07 | 0.0% | 2.3E-06 | 0.3% | 2.3E-06 | 0.0% |
| | | 1,2,3,7,8,9-HxCDF | 1.4E-07 | 0.0% | 1.4E-07 | 0.0% | 2.8E-07 | 0.0% |
| | | 2,3,4,6,7,8-HxCDF | 6.1E-08 | 0.0% | 6.1E-07 | 0.1% | 1.8E-06 | 0.0% |
| | | 1,2,3,4,6,7,8-HpCDF | 3.9E-06 | 0.6% | 7.7E-07 | 0.1% | 2.3E-05 | 0.1% |
| | | 1,2,3,4,7,8,9-HpCDF | 2.2E-08 | 0.0% | 1.1E-08 | 0.0% | 4.4E-08 | 0.0% |
| | | OCDF | 1.2E-10 | 0.0% | 1.2E-09 | 0.0% | 8.0E-09 | 0.0% |
| | | PCB-77 | 6.4E-07 | 0.1% | 3.2E-06 | 0.4% | 3.2E-03 | 16.9% |
| | | PCB-81 | 8.4E-07 | 0.1% | 4.2E-07 | 0.1% | 2.8E-05 | 0.1% |
| | | PCB-105 | 8.6E-07 | 0.1% | 5.1E-06 | 0.6% | 3.4E-04 | 1.8% |
| | | PCB-114 | 2.9E-06 | 0.5% | 4.3E-07 | 0.1% | 2.9E-05 | 0.2% |
| | | PCB-118 | 1.5E-06 | 0.2% | 2.3E-05 | 2.7% | 1.5E-03 | 8.1% |
| | | PCB-123 | 1.3E-07 | 0.0% | 4.0E-07 | 0.0% | 5.4E-06 | 0.0% |
| | | PCB-126 | 1.6E-05 | 2.6% | 1.6E-04 | 18.9% | 6.4E-04 | 3.4% |
| | | PCB-156/157 | 6.1E-06 | 1.0% | 1.8E-06 | 0.2% | 1.2E-02 | 64.8% |
| | | PCB-167 | 1.3E-07 | 0.0% | 7.7E-07 | 0.1% | 1.0E-05 | 0.1% |
| | | PCB-169 | 1.3E-07 | 0.0% | 5.4E-06 | 0.6% | 9.0E-05 | 0.5% |
| | | PCB-189 | 2.2E-08 | 0.0% | 1.3E-07 | 0.0% | 4.4E-07 | 0.0% |
| | | **Non-DL PCBs** | | | | | | |

| Angler (Adult + Child) RME Risk from Consumption of Crab (hepatopancreas + muscle) Based on Various TEFs ($TEF_{iL}$, $TEF_{WHO}$, and $TEF_{iU}$) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Receptor** | **Expo Pathway** | **Chemical of Potential Concern** | **$TEF_{iL}$** | | **$TEF_{WHO}$** | | **$TEF_{iU}$** | |
| | | | **Risk** | **Contribution to Total Risk (%)** | **Risk** | **Contribution to Total Risk (%)** | **Risk** | **Contribution to Total Risk (%)** |
| | | Total Non-DL PCBs | 7.0E-05 | 11.4% | 7.0E-05 | 8.4% | 7.0E-05 | 0.4% |
| | | **Total DLCs** | 5E-04 | 77.2% | 7E-04 | 83.2% | 2E-02 | 99.3% |
| | | **Total PCDD/Fs** | 4E-04 | 72.5% | 5E-04 | 59.4% | 6E-04 | 3.4% |
| | | **Total DL-PCBs** | 3E-05 | 4.7% | 2E-04 | 23.8% | 2E-02 | 95.9% |
| | | **Total PCBs (DL & NDL)** | 1E-04 | 16.1% | 3E-04 | 32.2% | 2E-02 | 96.2% |
| | | **TOTAL - All potential COCs** | 6E-04 | 100.0% | 8E-04 | 100.0% | 2E-02 | 100.0% |

### 7.3.4 Potential Contribution from Early-life Exposures to Lifetime Risk

The BHHRA addresses the potential increased susceptibility associated with early-life exposure to mutagens (limited to carcinogenic PAHs, trichloroethene, and hexavalent chromium) via use of ADAFs in the cancer risk calculations. Accordingly, all life stages potentially affected are addressed except pre-conception, in utero, and infant (0 to 1 year of age), because the youngest receptor evaluated is a child 1 to <7 years of age. If pregnant or breastfeeding mothers consume NBSA fish or crab, it is possible that an unborn child or nursing infant could be exposed to lipophilic and/or bioaccumulative potential COCs (e.g., PCDD/Fs, PCBs, mercury). The extent to which such women consume NBSA fish or crab is unknown.

### 7.3.5 Use of Surrogate Values

In several cases, surrogate chemicals (i.e., chemicals that are structurally similar) were used to evaluate risks/hazards from chemicals for which toxicity criteria from USEPA or other approved sources (e.g., ATSDR) are lacking. The use of surrogate toxicity criteria is often necessary, because toxicity information is not available for every chemical detected at complex sites such as the NBSA. The surrogates used in this BHHRA are consistent with those approved for use in the LPRSA BHHRA (AECOM 2017), including several recommended by USEPA's Superfund Health Risk Technical Support Center (STSC), and those recommended in the Revised PAR (Battelle 2018) and subsequent correspondence (USEPA 2018a). The use of toxicity criteria for structurally similar chemical surrogates may over- or underestimate the risk or hazard posed by a COPC lacking such data; however, the overall impact to the BHHRA conclusions is expected to be small.

### 7.3.6 Tier 3 Toxicity Values

USEPA specifies a level of confidence in Tier 1 (IRIS) and Tier 2 (STSC PPRTV) reference doses of low (e.g., antimony), medium (e.g., benzo(a)pyrene, PCBs) or high (e.g., 2,3,7,8-TCDD). There is additional

uncertainty associated with Tier 3 toxicity criteria because of variability in peer review or absence of consensus among the scientific community. The majority of the COPCs in the BHHRA have Tier 1 or Tier 2 toxicity criteria; however, Tier 3 criteria had to be identified for 10 COPCs, as summarized in the following table.

| COPC | Exposure Media | Tier 3 Toxicity Value | Type | Uncertainty Factor | Source |
|---|---|---|---|---|---|
| 4,4'-DDD (used as surrogate for 2,4'-DDD) | Surface water, biota | 3E-05 mg/kg-day | Oral RfD | 300 | USEPA PPRTV screening provisional value (USEPA 2017e) |
| 4,4'-DDE | Surface water, biota | 3E-04 mg/kg-day | Oral RfD | 3000 | USEPA PPRTV screening provisional value (USEPA 2017f) |
| Organic arsenic | Biota | 2E-02 mg/kg-day | Oral RfD | 100 | ATSDR MRL for dimethylarsinic acid (DMA) (ATSDR 2007) |
| Copper | Sediment, biota | 1E-03 mg/kg-day | Oral RfD | 30 | ATSDR intermediate MRL, (UF of 3) with additional UF of 10 applied (ATSDR 2004, USEPA and NJDEP 2019) |
| Thallium | Sediment, surface water | 1E-05 mg/kg-day | Oral RfD | 3000 | USEPA PPRTV screening provisional value (USEPA 2012e) |
| 2,3,7,8-TCDD | Sediment, surface water, biota | 150,000 $(mg/kg\text{-}day)^{-1}$ | Oral CSF | -- | HEAST (USEAP 1997a) |
| Chloroform | Surface water | 3.1E-02 $(mg/kg\text{-}day)^{-1}$ | Oral CSF | | CalEPA (2011) |
| Hexavalent chromium | Sediment, surface water | 0.5 $(mg/kg\text{-}day)^{-1}$ | Oral CSF | -- | NJDEP (2009) |
| Mirex | Biota | 1.8E-01 $(mg/kg\text{-}day)^{-1}$ | Oral CSF | -- | CalEPA (1992) |

Of these chemicals, only 2,4'-DDD, 4,4'-DDD, 4,4'-DDE, copper and 2,3,7,8-TCDD (and other DLCs) are identified as potential COCs in fish and crab tissue, which are discussed further below. For the remaining chemicals, the increased uncertainty associated with the use of Tier 3 toxicity criteria does not affect the overall conclusions of the BHHRA (i.e., estimated cancer risks were less than $10^{-6}$, and noncancer HIs were less than 0.1 for any receptor with a cumulative risk greater than $10^{-4}$ or HI greater than 1).

PPRTV screening values are used as the RfDs for several potential COCs. As discussed in Section 5.2, PPRTV screening values are less certain than standard PPRTVs but use current USEPA methodology in their derivation and undergo external peer review. However, these RfDs may be of limited utility to risk assessors as there is considerable uncertainty associated with these values (USEPA 2019a, 2019d).

Therefore, the substantial uncertainty associated with the PPRTV screening RfDs yields less certain hazards for these chemicals. This additional uncertainty is important for informing risk management decisions.

The oral RfD for 4,4'-DDD (which was also used as a surrogate for 2,4'-DDD) is a screening provisional value taken from the PPRTV appendix document (USEPA 2017e). The total UF is 300, and the critical effect is the liver. For 4,4'-DDD, the hazard quotient for the RME child angler/sportsman is 2 assuming consumption of a mixed fish diet, and 0.5 assuming consumption of a crab muscle and hepatopancreas diet. Accordingly, 4,4'-DDD may be unnecessarily identified as a COPC if the actual toxicity is lower than the current estimate. 2,4'-DDD was identified as a potential COPC in fish (not in crab); the hazard quotient for the RME child angler/sportsman is 0.4 assuming consumption of a mixed fish diet. Similarly, 2,4'-DDD may be unnecessarily identified as a potential COC if the actual toxicity is lower than the current estimate.

The oral RfD for 4,4'-DDE is a screening provisional value taken from the PPRTV appendix document (USEPA 2017f). The total UF is 3000, and the critical effect is the liver. The hazard quotient for the RME child angler/sportsman is 0.3 assuming consumption of a mixed fish diet, and 0.1 assuming consumption of a crab muscle and hepatopancreas diet. 4,4'-DDE may be unnecessarily identified as a potential COC if the actual toxicity is lower than the current estimate.

The oral RfD for copper is the ATSDR intermediate MRL with an additional UF of 10 applied, as recommended by USEPA (USEPA and NJDEP 2019). The intermediate MRL is based on a 2003 study in humans who ingested copper sulfate in drinking water for 2 months. The critical effect is GI effects. The HQ for the RME child angler/sportsman is 0.3 assuming consumption of a mixed fish diet, and 12 assuming consumption of a crab muscle and hepatopancreas diet. As discussed in Section 5.1, the Superfund Toxicity Workgroup recommends application of an UF between 3 and 10. USEPA Region 2 recommends 10 be used and the impacts of using an UF of 10 versus 3 be evaluated in the uncertainty evaluation (USEPA and NJDEP 2019), If an UF of 3 instead of 10 were applied, the resulting RfD would be approximately 3.3-fold higher (0.003 mg/kg-day instead of 0.001 mg/kg-day), yielding noncancer HQs approximately 3.3-fold lower (3.6 instead of 12 for RME child angler/sportsman consuming crab muscle and hepatopancreas, which is still higher than the goal of protection of a HQ of 1). As copper currently contributes to approximately 26% of the hazard for the RME child angler/sportsman consumption of crab muscle and hepatopancreas, if the copper RfD were reduced by as much as 3.3-fold, copper would continue to be a potential COC (HQ of 3.6), but it would contribute less to the hazard and the HI would be reduced (38 versus 46).

The oral CSF for 2,3,7,8-TCDD is 150,000 (mg/kg-day)-1 from HEAST (1997a), which is used as the reference point (along with congener-specific TEFs) for other PCDD/Fs and DL-PCBs. This value was used in the LPRSA BHHRA (AECOM 2017) and specified in the Revised PAR (Battelle 2018). This compound, along with other PCDD/Fs and DL-PCBs, is a major contributor to the cumulative cancer risk associated with consumption of NBSA fish or crab. Other Tier 3 toxicity criteria for 2,3,7,8-TCDD include 156,000 (mg/kg-day)$^{-1}$ (USEPA 1985) and 130,000 (mg/kg-day)$^{-1}$ (CalEPA 2011). These values are sufficiently similar so as to not affect the overall conclusions of the BHHRA.

## 7.4 Risk Characterization

As discussed in Section 6, risk characterization combines estimates of exposure and dose-response relationships to assess the potential for adverse health effects. There are several ways to introduce uncertainty into the risk characterization process, including simultaneous exposure to multiple chemicals, combination of multiple upper-bound assumptions, consideration of sensitive populations, and potential for risk from background (not site-related) exposures. Each of these issues is discussed in the following sections.

### 7.4.1 Risk from Multiple Chemicals

Estimated potential cancer risks are first estimated for each COPC individually, and then summed to estimate the cumulative cancer risk for each receptor (USEPA 1989). If two or more of the COPCs act synergistically (the combined effect is greater than additivity) or antagonistically (the combined effect is less than additivity), then the potential cancer risk may be over- or underestimated, respectively. There is relatively little information regarding interactions among groups of chemicals; however, because adding the risks for multiple carcinogenic chemicals assumes that all of the chemicals affect the same target organ by the same mode of action, the assumption of additivity is likely reasonably conservative. Additivity also ignores that individual slope factors reflect upper-bound estimates of potency, and therefore, are not directly additive. Furthermore, adding risks across all carcinogenic chemicals also ignores that there are varying levels of evidence of carcinogenicity in humans. In fact, only a relatively few of the carcinogenic COPCs are considered known human carcinogens. In total, the current method of summing cancer risks across multiple carcinogenic chemicals is unlikely to underestimate the total risk.

Estimated noncancer hazards are also first estimated for each COPC individually, and then summed to estimate the cumulative HI for each receptor (USEPA 1989). In addition, separate HIs are calculated based on target endpoint (e.g., reproductive effects), recognizing that an individual chemical may cause more than one effect (e.g., inorganic arsenic can adversely affect the skin and blood). The uncertainty in this approach is unknown; however, in this assessment, either at least one chemical had an HI greater than 1, or all chemicals had HIs below 1 and the sum was also below 1.

### 7.4.2 Combination of Several Upper-Bound Assumptions

As discussed in Section 4, consistent with USEPA guidance (USEPA 1992a), two exposure scenarios are evaluated in the BHHRA that represent the reasonable maximum exposure (RME) and central tendency exposure (CTE). One combination of assumptions evaluated is meant to be representative of RME that is above the average case but within the range of reasonably possible exposures (USEPA 1992a). Exposure parameters that are variable or uncertain are selected to be a mix of average and higher-end values within their ranges, avoiding the unrealistically high exposure estimate that would result from using all upper-bound or maximum values (USEPA 1989). Another combination is meant to be representative of CTE that is the average level of exposure predicted for the receptors (USEPA 1992a). This estimate is developed by assuming average or central tendency values for most or all exposure assumptions.

A number of the values used in the BHHRA are standard default exposure parameter assumptions recommended by USEPA for Superfund site human health risk assessments (USEPA 1989, 1991b, 2004b, 2011, 2012c, 2014). These default assumptions for the RME scenarios were developed by USEPA, in many cases based on large amounts of data, to be used in combination to represent a person (within certain age groups) experiencing the upper range of possible exposures. For example, for the predominant contributing pathway to risks, fish and crab ingestion, the mix of upper-bound and average assumptions used in this BHHRA for RME calculations includes:

- 90$^{th}$ percentile fish and crab consumption rate
- 95% upper confidence limit on the arithmetic mean concentrations of chemicals in fish and crab tissue
- No loss of chemicals from the fish or crab tissue due to cooking or consumption practices (i.e., the upper-bound assumption that fat and cooking juices are always consumed)
- All of the fish/crab consumed comes from the NBSA
- 90$^{th}$ percentile exposure duration
- Mean body weight
- Upper-bound cancer slope factors.

Use of this mix of assumptions results in estimates of potential risk that are likely to be well above the risk that may be experienced by receptors in the NBSA.

#### 7.4.3 Risks to Sensitive Populations

Variability within the human population is inevitable, with some people being more sensitive to chemical exposures than others. Accordingly, dose-response values used to estimate risk (cancer slope factors and noncancer reference doses) are generally derived to account for sensitive subpopulations. For example, in both cases, the dose-response value is generally based on the most sensitive species and most sensitive endpoint. In addition, cancer slope factors represent upper-bound values, whereas reference doses routinely include an uncertainty factor of 10 to account for intraspecies differences. Finally, ADAFs were applied to the estimation of cancer risk from mutagenic compounds to account for the potential increased risk of early-life exposures. In total, these assumptions are intended to be protective of the vast majority of the human population.

### 7.5 Summary of Uncertainty in BHHRA for the NBSA

The evaluation of uncertainty inherent to the BHHRA explains how assumptions used for exposure concentrations, exposure factors, and toxicity factors account for uncertainties in a manner that provides confidence that the BHHRA overestimates rather than underestimates actual risks, particularly for the RME scenarios. In general, this confidence in the conservatism in RME exposure and risk estimates derives from the use of a mix of:

- The lesser of the 95% upper confidence limit of the mean or maximum detected potential COC concentration for the exposure-point concentrations
- Largely high-end and some average exposure factors

- High-end toxicity factors.

The discussion of uncertainties also improves the transparency and understanding of assumptions used in the BHHRA. These RME estimates, along with CTE estimates for perspective, provide useful information about the reliability of the BHHRA results for risk management decision making.

# 8. Summary and Conclusions

This BHHRA has been conducted as part of the RI/FS for the NBSA to address current and reasonably foreseeable future uses in the absence of controls or remedial actions (i.e., "baseline" conditions). The BHHRA has been performed in a manner consistent with the Revised Pathways Analysis Report (Revised PAR) for the NBSA (Battelle 2018), including receptors and exposure pathways evaluated and exposure assumptions used for both RME and CTE scenarios. In addition, the BHHRA addresses comments and revisions provided by USEPA, USEPA review of responses to comments, and agreed-upon resolutions (USEPA 2017a, 2017b, 2017c, 2018a, 2018b, 2018c).

## 8.1 Summary of BHRRA for the NBSA

The BHHRA was conducted in accordance with USEPA's four-step risk assessment paradigm (USEPA 1989):

- Data evaluation and hazard identification
- Exposure assessment
- Toxicity assessment
- Risk characterization.

Each of the four steps is summarized below.

### 8.1.1 Data Evaluation and Hazard Identification

The BHHRA was based solely on validated data from the RI/FS program, which were collected in accordance with Quality Assurance Project Plans (QAPPs) approved by USEPA Region 2. These include:

- 41 accessible surface sediment samples (including field duplicates) from 39 nearshore and mudflat locations
- 131 near-surface (shallow) surface water samples from six locations in Newark Bay
- 95 samples (including duplicates) from five fish species (American eel, bluefish, striped bass, summer flounder, and white perch)
- 37 samples each of crab muscle only and crab hepatopancreas only.

All data were validated according to approved QAPPs, with nearly all of the data determined to be valid and acceptable for use in the BHHRA, as qualified. A total of 84 chemicals were identified as potential chemicals of concern (potential COCs) in one or more of these media based on a screening process that considered carcinogen status, essential nutrient status, frequency of detection, and comparison of maximum concentrations to risk-based screening levels, consistent with the Revised PAR. These included polychlorinated dibenzo(p)dioxins and furans (PCDD/Fs), polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs), various pesticides and inorganics, and a few total petroleum hydrocarbon (TPH) ranges, volatile organic compounds (VOCs) and semivolatile organic compounds (SVOCs). An

additional 56 chemicals were evaluated qualitatively in the uncertainty evaluation. The potential COC screening process was designed to ensure that chemicals not identified as potential COCs are only minor contributors to overall site risks and noncancer hazards.

### 8.1.2 Exposure Assessment

Newark Bay is central to one of the most urbanized and industrialized areas in the United States. Human use of the NBSA is primarily industrial and commercial. Recreational use is limited by access impediments from the shoreline (i.e., bulkhead, bridges, sheet piling, and mudflats) and surrounding urban/industrial/commercial land use. Access for recreation is gained through available public access areas and pleasure boating (i.e., from marinas inside and outside of the NBSA).

Potential receptors and exposure pathways identified for quantitative evaluation in the HHCSM for the NBSA include the following:

- Angler/sportsmen who may be exposed via fish or shellfish ingestion, dermal contact with sediment and surface water, and incidental ingestion of sediment and surface water
- Swimmers, waders, and boaters who may be exposed via dermal contact with sediment and surface water, and incidental ingestion of sediment and surface water
- Workers who may be exposed via dermal contact with sediment and incidental ingestion of sediment.

Potential exposure via inhalation of vapors in outdoor air was excluded from the final cumulative risk estimates based on the results of a quantitative screening-level evaluation that showed negligible risks to all receptors. Other pathways not included were ingestion of waterfowl or species other than fish and crabs, and potential exposure by residential or transient receptors, because these potential exposures are expected to be less than that experienced by the receptors included in the quantitative risk assessment.

The BHHRA included evaluation of both an RME and CTE scenario to provide an estimate of the range of risks of the exposed population, even though decisions at Superfund sites are traditionally based on the RME scenario. The fish and crab ingestion rates established by USEPA Region 2 for the Lower Passaic River Study Area (LPRSA) were used in this BHHRA. Exposure to fish and crab tissue, as well as accessible surface sediment and surface water, is evaluated on a sitewide (Bay-wide) basis. In addition, the exposure-point concentration for both the RME and CTE scenarios is the lower of the 95 percent upper confidence limit (95% UCL) of the arithmetic mean or maximum concentration, consistent with USEPA guidance.

The BHHRA evaluated a “mixed fish” diet to account for the presence of multiple fish species in Newark Bay that may be consumed by anglers, which is assumed to be composed of equal amounts (20%) of the five species collected as part of the RI/FS (American eel, bluefish, striped bass, summer flounder, and white perch). A supplemental analysis of individual fish species diets was included in the uncertainty evaluation. Similarly, the BHHRA evaluated crab muscle and hepatopancreas tissues combined, to account for the possibility that the crab is cooked before the hepatopancreas is removed. A supplemental analysis of a crab-

muscle-only diet was included in the uncertainty evaluation (Section 7). Finally, no cooking loss is considered in the RME scenario for both fish and crab consumption, which assumes that fat, pan drippings, and cooking juices are consumed. For the CTE scenario, cooking loss was included for fish consumption (insufficient data are available for crab consumption).

### 8.1.3 Toxicity Assessment

The toxicity criteria used in the BHHRA were selected according to USEPA (2003a, 2019b) guidance, including cancer and noncancer criteria for oral and dermal exposures. USEPA (2004b) default dermal absorption factors were used to adjust oral toxicity criteria for evaluating dermal exposure. In addition, USEPA's age-dependent adjustment factors were used to evaluate early-life exposures for chemicals believed to act by a mutagenic mode of action (USEPA 2005c). Blood lead models were used to evaluate potential exposure to lead (USEPA 1994a, 1994b, 2017d; Bowers et al. 1994).

For PCDD/Fs and dioxin-like (DL) PCBs, cancer risks and hazard indices were estimated for the individual congeners, as well as in terms of a total toxicity equivalence (TEQ) for PCDD/Fs and PCBs (TEQ DF and TEQ PCB, respectively). The toxicity criteria for these compounds are based on the cancer and noncancer criteria for 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) and congener-specific toxicity equivalency factors (TEFs). The TEQ DF and TEQ PCB were calculated by two methods: (1) using USEPA's Kaplan-Meier (KM) calculator (Version 9.1; issued July 2014), and (2) manually, based on the TEQ concentration for each congener. The remaining non-DL PCB congeners were evaluated as a group (Total non-DL PCBs) using toxicity criteria for PCBs (high risk) and Aroclor 1254 for cancer and noncancer effects, respectively. Cumulative risk/hazard estimates are presented based on KM TEQs (denoted "Total (based on KM TEQs)," as well as based on TEQs calculated manually (denoted "Total (excluding KM TEQs)." The calculations were performed both ways in order to employ the KM TEQ calculator [as was done for the LPRSA BHHRA (AECOM 2017)], allow for determination of risks/hazards for individual congeners via the manual calculations, and then compare the results of the two methods. As discussed further below, there is essentially no difference in the risk/hazard estimates between the two methods; however, the latter method allows for identification of the specific congeners that contribute most to the overall risk/hazard.

### 8.1.4 Risk Characterization

The estimated cancer risks were compared to the NCP risk range of $10^{-6}$ to $10^{-4}$, and estimated noncancer hazards were compared to a hazard index of 1 (USEPA 1991d). In addition, noncancer hazard indices greater than 1 were evaluated further on a target-organ-specific basis (USEPA 1989). Consumption of NBSA fish or crab by an angler/sportsman represents the only exposure pathways for which estimated potential cancer risks are above the NCP risk range of $10^{-6}$ to $10^{-4}$ and noncancer HIs are above 1 (USEPA 1991d). Estimated cancer risks/hazard associated with direct contact with accessible surface sediment and surface water are below the NCP risk range and noncancer protection goal for all receptors. The results of the risk characterization are summarized in the following sections, as well as in Figure 8-1 (cumulative cancer risks) and Figures 8-2 through 8-6 (cumulative noncancer HIs for five target organs with HIs greater than 1).

*8.1.4.1 Fish Consumption*

The cumulative potential cancer risk for the RME combined adult/child angler/sportsman who routinely consumes a mixed diet of self-caught fish over a period of 26 years is $8\times10^{-4}$, regardless of TEQ approach, as shown in the summary table below. The primary contributors to the RME cumulative potential cancer risks are 2,3,7,8-TCDD, which contributes approximately 28% ($2\times10^{-4}$) (33% or 34% for all PCDD/Fs, depending on TEQ approach [$3\times10^{-4}$]), PCB-126, which contributes approximately 31% ($2\times10^{-4}$) (36% or 38% for all DL-PCBs, depending on TEQ approach [$3\times10^{-4}$]), and non-DL PCBs, which contributes approximately 18% or 19%, depending on TEQ approach ($1\times10^{-4}$).[7] Minor contributors to the cumulative cancer risk include pesticides (approximately 5% [$4\times10^{-4}$]) and inorganic arsenic (approximately 4% [$3\times10^{-5}$]). Potential cancer risks associated with direct contact with accessible surface sediment or surface water are within or below the NCP risk range for the RME scenario.

**Summary of Angler/Sportsman Fish Consumption Cancer Risk and Percent Contribution to Cumulative Risk for Potential COCs**

**RME Adult/Child Angler/Sportsman — Consumption of Mixed Fish Diet**

| Potential COC | Cancer Risk | | | | | |
|---|---|---|---|---|---|---|
| | Accessible Surface Sediment (a) | Surface Water (a) | Mixed Fish Diet (b) | Total Potential Risk | Percent Contribution to Cumulative Risk (excluding KM TEQ) (c)(d)(e) | Percent Contribution to Cumulative Risk (based on KM TEQ) |
| 2,3,7,8-TCDD | 6E-07 | 9E-10 | 2E-04 | 2E-04 | 28% | -- |
| 1,2,3,7,8-PeCDD | 3E-08 | 3E-10 | 1E-05 | 1E-05 | 2% | -- |
| 1,2,3,6,7,8-HxCDD | 1E-08 | 4E-11 | 2E-06 | 2E-06 | 0.2% | -- |
| 2,3,7,8-TCDF | 1E-08 | 4E-11 | 5E-06 | 5E-06 | 1% | -- |
| 1,2,3,7,8-PeCDF | 2E-09 | 9E-12 | 1E-06 | 1E-06 | 0.2% | -- |
| 2,3,4,7,8-PeCDF | 4E-08 | 2E-10 | 2E-05 | 2E-05 | 3% | -- |
| 1,2,3,4,7,8-HxCDF | 6E-08 | 1E-10 | 3E-06 | 3E-06 | 0.4% | -- |
| 1,2,3,6,7,8-HxCDF | 1E-08 | 3E-11 | 3E-06 | 3E-06 | 0.3% | -- |
| Total PCDD/Fs (excluding KM TEQ) | 8E-07 | 2E-09 | 3E-04 | 3E-04 | 34% | -- |
| Total PCDD/Fs (based on KM TEQ) | 8E-07 | 2E-09 | 3E-04 | 3E-04 | -- | 33% |
| PCB-77 | 6E-09 | 2E-12 | 1E-06 | 1E-06 | 0.2% | -- |
| PCB-105 | 3E-09 | 2E-12 | 6E-06 | 6E-06 | 0.8% | -- |
| PCB-118 | 8E-09 | 4E-12 | 3E-05 | 3E-05 | 3.2% | -- |
| PCB-126 | 2E-07 | 5E-11 | 2E-04 | 2E-04 | 31% | -- |
| PCB-156/157 | 7E-10 | 5E-13 | 2E-06 | 2E-06 | 0.3% | -- |
| PCB-167 | 3E-10 | 2E-13 | 1E-06 | 1E-06 | 0.1% | -- |
| PCB-169 | 6E-08 | 9E-12 | 3E-06 | 3E-06 | 0.4% | -- |
| Total DL-PCBs (excluding KM TEQ) | 3E-07 | 7E-11 | 3E-04 | 3E-04 | 36% | -- |
| Total DL-PCBs (based on KM TEQ) | 2E-07 | 8E-11 | 3E-04 | 3E-04 | -- | 38% |

[7] The percentages presented in this section may be slightly different from those presented in Section 6, because the small contributions from potential exposure to accessible surface sediment and surface water are included.

**Summary of Angler/Sportsman Fish Consumption Cancer Risk and Percent Contribution to Cumulative Risk for Potential COCs**

**RME Adult/Child Angler/Sportsman — Consumption of Mixed Fish Diet**

| Potential COC | Cancer Risk | | | | | |
|---|---|---|---|---|---|---|
| | Accessible Surface Sediment (a) | Surface Water (a) | Mixed Fish Diet (b) | Total Potential Risk | Percent Contribution to Cumulative Risk (excluding KM TEQ) (c)(d)(e) | Percent Contribution to Cumulative Risk (based on KM TEQ) |
| Total Non-DL PCBs | 2E-07 | -- | 1E-04 | 1E-04 | 19% | 18% |
| Benzo(a)pyrene | 3E-07 | 8E-11 | 3E-06 | 3E-06 | 0.4% | 0.4% |
| Dibenz(a,h)anthracene | 6E-08 | 2E-11 | 3E-06 | 3E-06 | 0.4% | 0.4% |
| 2,4'-DDD | -- | -- | 8E-07 | 8E-07 | 0.1% | 0.1% |
| 4,4'-DDD | -- | -- | 4E-06 | 4E-06 | 0.5% | 0.5% |
| 4,4'-DDE | -- | -- | 8E-06 | 8E-06 | 1% | 1% |
| Chlordane, alpha (cis) | -- | -- | 1E-06 | 1E-06 | 0.1% | 0.1% |
| Dieldrin | -- | -- | 2E-05 | 2E-05 | 3% | 3% |
| Heptachlor epoxide, cis- | -- | -- | 3E-06 | 3E-06 | 0.4% | 0.4% |
| Heptachlor epoxide, trans- | -- | -- | 1E-07 | 1E-07 | 0.02% | 0.02% |
| Nonachlor, trans- | -- | -- | 1E-06 | 1E-06 | 0.1% | 0.1% |
| Arsenic, inorganic | 2E-06 | 2E-08 | 3E-05 | 3E-05 | 4% | 4% |
| **Total Potential Risk (excluding KM TEQs) (f)(g)** | **4E-06** | **5E-08** | **8E-04** | **8E-04** | **99%** | **--** |
| **Total Potential Risk (with KM TEQs) (f)(g)** | **4E-06** | **5E-08** | **8E-04** | **8E-04** | **--** | **99%** |

Notes:
Highlighting indicates potential risk exceeding the NCP risk range of 1E-6 to 1E-4.
(a) Adult age group. Child angler is not assumed to be exposed to sediment or surface water.
(b) Combined adult/child.
(c) Total PCDD/Fs (excluding KM TEQ)—contains contribution of all PCDD/F congeners.
(d) Total DL-PCBs (excluding KM TEQ)—contains contribution of all DL-PCB congeners.
(e) Individual congener percentage contributions were not included in total percent cumulative risk.
(f) Includes risks posed by other potential COCs not shown in table.
(g) The sum of percent contribution may not be the same as when the individual values are summed due to rounding.

The cumulative potential noncancer HIs for the RME child angler who routinely consumes fish from the NBSA is 40, regardless of TEQ approach, as shown in the summary table below. As with excess cancer risk, the primary contributors to the cumulative potential HIs are 2,3,7,8-TCDD, which contributes approximately 19% (HI of 8) (22% or 23% for all PCDD/Fs, depending on TEQ approach [HI of 10]), PCB-126, which contributes approximately 20% (HI of 9) (24% to 26% for all DL-PCBs, depending on TEQ approach [HI of 10]), and non-DL PCBs, which contribute approximately 32% or 33%, depending on TEQ approach (HI of 10). The highest target-organ-specific HI is 20 for reproductive effects (DLCs), regardless of TEQ approach. The next-highest target-organ-specific HI is 10 for whole-body effects (non-DL PCBs), regardless of TEQ approach. Liver (pesticides) and neurological effects (methyl mercury) are the only other target-organ-specific HIs greater than one (5 and 2, respectively).

**Summary of Angler/Sportsman Fish Consumption Noncancer Hazard and Percent Contribution to Cumulative Hazards for Potential COCs**

**RME Child Angler/Sportsman — Consumption of Mixed Fish Diet**

| Primary Target Organ(s) | Potential COC | Noncancer Hazard | | | | | |
|---|---|---|---|---|---|---|---|
| | | Accessible Surface Sediment (a) | Surface Water (a) | Mixed Fish Diet | Total Hazard | Percent Contribution to Cumulative Hazard (excluding KM TEQ) (b)(c)(d) | Percent Contribution to Cumulative Hazard (based on KM TEQ) |
| Reproductive | 2,3,7,8-TCDD | -- | -- | 8E+00 | 8E+00 | 19% | -- |
| | 1,2,3,7,8-PeCDD | -- | -- | 4E-01 | 4E-01 | 1% | -- |
| | 1,2,3,6,7,8-HxCDD | -- | -- | 6E-02 | 6E-02 | 0.1% | -- |
| | 2,3,7,8-TCDF | -- | -- | 2E-01 | 2E-01 | 0.4% | -- |
| | 1,2,3,7,8-PeCDF | -- | -- | 4E-02 | 4E-02 | 0.1% | -- |
| | 2,3,4,7,8-PeCDF | -- | -- | 7E-01 | 7E-01 | 2% | -- |
| | 1,2,3,4,7,8-HxCDF | -- | -- | 1E-01 | 1E-01 | 0.2% | -- |
| | 1,2,3,6,7,8-HxCDF | -- | -- | 1E-01 | 1E-01 | 0.2% | -- |
| | Total PCDD/Fs (excluding KM TEQ) | -- | -- | 1E+01 | 1E+01 | 23% | -- |
| | Total PCDD/Fs (based on KM TEQ) | -- | -- | 1E+01 | 1E+01 | -- | 22% |
| | PCB-77 | -- | -- | 5E-02 | 5E-02 | 0.1% | -- |
| | PCB-105 | -- | -- | 2E-01 | 2E-01 | 1% | -- |
| | PCB-118 | -- | -- | 9E-01 | 9E-01 | 2% | -- |
| | PCB-126 | -- | -- | 9E+00 | 9E+00 | 20% | -- |
| | PCB-156/157 | -- | -- | 8E-02 | 8E-02 | 0.2% | -- |
| | PCB-167 | -- | -- | 4E-02 | 4E-02 | 0.1% | -- |
| | PCB-169 | -- | -- | 1E-01 | 1E-01 | 0.3% | -- |
| | Total DL-PCBs (excluding KM TEQ) | -- | -- | 1E+01 | 1E+01 | 24% | -- |
| | Total DL-PCBs (based on KM TEQ) | -- | -- | 1E+01 | 1E+01 | -- | 26% |
| Whole Body | Total Non-DL PCBs | -- | -- | 1E+01 | 1E+01 | 32% | 32% |
| Developmental | Benzo(a)pyrene | -- | -- | 2E-02 | 2E-02 | 0.04% | 0.04% |
| Liver | 2,4'-DDD | -- | -- | 4E-01 | 4E-01 | 1% | 1% |
| | 4,4'-DDD | -- | -- | 2E+00 | 2E+00 | 5% | 5% |
| | 4,4'-DDE | -- | -- | 3E-01 | 3E-01 | 1% | 1% |
| | Chlordane, alpha (cis) | -- | -- | 2E-02 | 2E-02 | 0.1% | 0.1% |
| | Dieldrin | -- | -- | 1E-01 | 1E-01 | 0.2% | 0.2% |
| | Heptachlor epoxide, cis- | -- | -- | 1E-01 | 1E-01 | 0.2% | 0.2% |
| | Nonachlor, trans- | -- | -- | 7E-01 | 7E-01 | 2% | 2% |
| | Pyridine | -- | -- | 9E-01 | 9E-01 | 2% | 2% |
| Skin, Blood | Arsenic, inorganic | -- | -- | 3E-01 | 3E-01 | 1% | 1% |
| Thyroid | Cobalt | -- | -- | 5E-01 | 5E-01 | 1% | 1% |
| GI Tract | Copper | -- | -- | 3E-01 | 3E-01 | 1% | 1% |
| Immune | Mercury | -- | -- | 7E-01 | 7E-01 | 2% | 2% |
| Neurological | Methyl Mercury | -- | -- | 2E+00 | 2E+00 | 6% | 6% |
| | **Total Hazard (excluding KM TEQ) (e)(f)** | **--** | **--** | **4E+01** | **4E+01** | **98%** | **--** |
| | **Total Hazard (based on KM TEQ) (e)(f)** | **--** | **--** | **4E+01** | **4E+01** | **--** | **99%** |

Notes:
Highlighting indicates that the hazard exceeds the goal of protection of a hazard index of one.
(a) Child angler is not assumed to be exposed to sediment or surface water.

**Summary of Angler/Sportsman Fish Consumption Noncancer Hazard and Percent Contribution to Cumulative Hazards for Potential COCs**

**RME Child Angler/Sportsman — Consumption of Mixed Fish Diet**

| Primary Target Organ(s) | Potential COC | Noncancer Hazard | | | | | |
|---|---|---|---|---|---|---|---|
| | | Accessible Surface Sediment (a) | Surface Water (a) | Mixed Fish Diet | Total Hazard | Percent Contribution to Cumulative Hazard (excluding KM TEQ) (b)(c)(d) | Percent Contribution to Cumulative Hazard (based on KM TEQ) |

(b) Total PCDD/Fs (excluding KM TEQ)—contains contribution of all PCDD/F congeners.
(c) Total DL-PCBs (excluding KM TEQ)—contains contribution of all DL-PCB congeners.
(d) Individual congener percentage contributions were not included in total percent cumulative hazard.
(e) Includes hazard posed by other potential COCs not shown in table.
(f) The sum of percent contribution may not be the same as when the individual values are summed due to rounding.

The cumulative potential cancer risks for the CTE scenario for mixed fish diet are within the NCP risk range. For noncancer HIs, the only CTE target organ-specific HI greater than 1 (USEPA 1991d) is for reproductive effects (DLCs), where the HI is 2, regardless of TEQ approach.

*8.1.4.2 Crab Consumption*

The cumulative potential cancer risk for the RME combined adult/child angler/sportsman who routinely consumes a diet of self-caught crab muscle and hepatopancreas over a period of 26 years is also $8\times10^{-4}$, regardless of TEQ approach, as shown in the summary table below. The primary contributors to the RME cumulative potential cancer risks are 2,3,7,8-TCDD, which contributes approximately 52% ($4\times10^{-4}$) (59% or 60% for all PCDD/Fs, depending on TEQ approach [$5\times10^{-4}$]), PCB-126, which contributes approximately 19% ($2\times 10^{-4}$) (23% or 24% for all DL-PCBs, depending on TEQ approach [$2\times10^{-4}$]), and non-DL PCBs, which contributes approximately 8%, regardless of TEQ approach ($7\times10^{-5}$). Minor contributors to the cumulative cancer risk include inorganic arsenic (approximately 6% [$5\times10^{-5}$]) and pesticides (approximately 3% [$2\times10^{-5}$]). Potential cancer risks associated with direct contact with accessible surface sediment or surface water are within or below the NCP risk range for the RME scenario.

**Summary of Angler/Sportsman Crab Consumption Cancer Risk and Percent Contribution to Cumulative Risk for Potential COCs**

**RME Adult/Child Angler/Sportsman — Consumption of Crab Muscle and Hepatopancreas**

| Potential COC | Cancer Risk | | | | | |
|---|---|---|---|---|---|---|
| | Accessible Surface Sediment (a) | Surface Water (a) | Crab Muscle & Hepato (b) | Total Potential Risk | Percent Contribution to Cumulative Risk (excluding KM TEQ) (c)(d)(e) | Percent Contribution to Cumulative Risk (based on KM TEQ) |
| 2,3,7,8-TCDD | 4E-07 | 6E-10 | 4E-04 | 4E-04 | 52% | -- |
| 1,2,3,7,8-PeCDD | 2E-08 | 2E-10 | 1E-05 | 1E-05 | 1% | -- |
| 2,3,7,8-TCDF | 7E-09 | 2E-11 | 2E-05 | 2E-05 | 2% | -- |
| 1,2,3,7,8-PeCDF | 1E-09 | 6E-12 | 1E-06 | 1E-06 | 0.1% | -- |
| 2,3,4,7,8-PeCDF | 2E-08 | 1E-10 | 2E-05 | 2E-05 | 3% | -- |
| 1,2,3,4,7,8-HxCDF | 3E-08 | 9E-11 | 8E-06 | 8E-06 | 1% | -- |
| 1,2,3,6,7,8-HxCDF | 9E-09 | 2E-11 | 2E-06 | 2E-06 | 0.3% | -- |
| Total PCDD/Fs (excluding KM TEQ) | 5E-07 | 1E-09 | 5E-04 | 5E-04 | 59% | -- |
| Total PCDD/Fs (based on KM TEQ) | 5E-07 | 1E-09 | 5E-04 | 5E-04 | -- | 60% |
| PCB-77 | 4E-09 | 1E-12 | 3E-06 | 3E-06 | 0.4% | -- |
| PCB-105 | 2E-09 | 1E-12 | 5E-06 | 5E-06 | 1% | -- |
| PCB-118 | 5E-09 | 2E-12 | 2E-05 | 2E-05 | 3% | -- |
| PCB-126 | 1E-07 | 3E-11 | 2E-04 | 2E-04 | 19% | -- |
| PCB-156/157 | 5E-10 | 3E-13 | 2E-06 | 2E-06 | 0.2% | -- |
| PCB-169 | 4E-08 | 6E-12 | 5E-06 | 5E-06 | 1% | -- |
| Total DL-PCBs (excluding KM TEQ) | 2E-07 | 4E-11 | 2E-04 | 2E-04 | 24% | -- |
| Total DL-PCBs (based on KM TEQ) | 1E-07 | 5E-11 | 2E-04 | 2E-04 | -- | 23% |
| Total Non-DL PCBs | 1E-07 | -- | 7E-05 | 7E-05 | 8% | 8% |
| 4,4'-DDD | -- | -- | 9E-07 | 9E-07 | 0.1% | 0.1% |
| 4,4'-DDE | -- | -- | 4E-06 | 4E-06 | 0.4% | 0.4% |
| Dieldrin | -- | -- | 1E-05 | 1E-05 | 1% | 1% |
| Heptachlor epoxide, cis- | -- | -- | 4E-06 | 4E-06 | 0.4% | 0.4% |
| Heptachlor epoxide, trans- | -- | -- | 1E-06 | 1E-06 | 0.1% | 0.1% |
| Nonachlor, trans- | -- | -- | 4E-07 | 4E-07 | 0.05% | 0.05% |
| Arsenic, inorganic | 1E-06 | 1E-08 | 5E-05 | 5E-05 | 6% | 6% |
| **Total Potential Risk (excluding KM TEQs) (f)(g)** | **2E-06** | **3E-08** | **8E-04** | **8E-04** | **100%** | **--** |
| **Total Potential Risk (with KM TEQs) (f)(g)** | **2E-06** | **3E-08** | **8E-04** | **8E-04** | **--** | **100%** |

Notes:
Highlighting indicates potential risk exceeding the NCP risk range of 1E-6 to 1E-4
(a) Adult age group. Child angler is not assumed to be exposed to sediment or surface water.
(b) Combined adult/child
(c) Total PCDD/Fs (excluding KM TEQ)—contains contribution of all PCDD/F congeners.
(d) Total DL-PCBs (excluding KM TEQ)—contains contribution of all DL-PCB congeners.
(e) Individual congener percentage contributions were not included in total percent cumulative risk.
(f) Includes risks posed by other potential COCs not shown in table.
(g) The sum of percent contribution may not be the same as when the individual values are summed due to rounding.

The cumulative potential noncancer HIs for the RME child angler who routinely consumes muscle and hepatopancreas from the NBSA is 50, regardless of TEQ approach, as shown in the summary table below. As with excess cancer risk, the primary contributors to the cumulative potential HIs are 2,3,7,8-TCDD, which contributes approximately 33% (HI of 20) (38% for all PCDD/Fs, regardless of TEQ approach [HI of 20]), copper, which contributes approximately 25% or 26%, depending on TEQ approach (HI of 10), PCB-126, which contributes approximately 12% (HI of 6) (15% for all DL-PCBs, regardless of TEQ approach [HI of 7]), and non-DL PCBs, which contribute approximately 14%, regardless of TEQ approach (HI of 7). The highest target-organ-specific HI is 20 for reproductive effects (DLCs), regardless of TEQ approach. The next-highest target-organ-specific HIs are 10 for GI tract (copper) and 7 for whole-body effects (non-DL PCBs), regardless of TEQ approach. The remaining target-organ-specific HIs are equal to or less than the goal of 1 (USEPA 1991d).

**Summary of Angler/Sportsman Crab Consumption Noncancer Hazard and Percent Contribution to Cumulative Hazards for Potential COCs**

**RME Child Angler/Sportsman — Consumption of Crab Muscle and Hepatopancreas**

| Primary Target Organ(s) | Potential COC | Noncancer Hazard | | | | | |
|---|---|---|---|---|---|---|---|
| | | Accessible Surface Sediment (a) | Surface Water (a) | Crab Muscle & Hepato | Total Hazard | Percent Contribution to Cumulative Hazard (excluding KM TEQ) (b)(c)(d) | Percent Contribution to Cumulative Hazard (based on KM TEQ) |
| Reproductive | 2,3,7,8-TCDD | -- | -- | 2E+01 | 2E+01 | 33% | -- |
| | 1,2,3,7,8-PeCDD | -- | -- | 4E-01 | 4E-01 | 1% | -- |
| | 2,3,7,8-TCDF | -- | -- | 6E-01 | 6E-01 | 1% | -- |
| | 1,2,3,7,8-PeCDF | -- | -- | 4E-02 | 4E-02 | 0.1% | -- |
| | 2,3,4,7,8-PeCDF | -- | -- | 9E-01 | 9E-01 | 2% | -- |
| | 1,2,3,4,7,8-HxCDF | -- | -- | 3E-01 | 3E-01 | 0.6% | -- |
| | 1,2,3,6,7,8-HxCDF | -- | -- | 8E-02 | 8E-02 | 0.2% | -- |
| | Total PCDD/Fs (excluding KM TEQ) | -- | -- | 2E+01 | 2E+01 | 38% | -- |
| | Total PCDD/Fs (based on KM TEQ) | -- | -- | 2E+01 | 2E+01 | -- | 38% |
| | PCB-77 | -- | -- | 1E-01 | 1E-01 | 0.2% | -- |
| | PCB-105 | -- | -- | 2E-01 | 2E-01 | 0.4% | -- |
| | PCB-118 | -- | -- | 8E-01 | 8E-01 | 2% | -- |
| | PCB-126 | -- | -- | 6E+00 | 6E+00 | 12% | -- |
| | PCB-156/157 | -- | -- | 7E-02 | 7E-02 | 0.1% | -- |
| | PCB-169 | -- | -- | 2E-01 | 2E-01 | 0.4% | -- |
| | Total DL-PCBs (excluding KM TEQ) | -- | -- | 7E+00 | 7E+00 | 15% | -- |
| | Total DL-PCBs (based on KM TEQ) | -- | -- | 7E+00 | 7E+00 | -- | 15% |
| Whole Body | Total Non-DL PCBs | -- | -- | 7E+00 | 7E+00 | 14% | 19% |
| Liver | 4,4'-DDD | -- | -- | 5E-01 | 5E-01 | 1% | 1% |
| | 4,4'-DDE | -- | -- | 1E-01 | 1E-01 | 0.3% | 0.4% |
| | Dieldrin | -- | -- | 5E-02 | 5E-02 | 0.1% | 0.1% |
| | Heptachlor epoxide, cis- | -- | -- | 1E-01 | 1E-01 | 0.2% | 0.3% |
| | Heptachlor epoxide, trans- | -- | -- | 4E-02 | 4E-02 | 0.1% | 0.1% |
| | Nonachlor, trans- | -- | -- | 3E-01 | 3E-01 | 0.6% | 1% |

**Summary of Angler/Sportsman Crab Consumption Noncancer Hazard and Percent Contribution to Cumulative Hazards for Potential COCs**

**RME Child Angler/Sportsman — Consumption of Crab Muscle and Hepatopancreas**

| Primary Target Organ(s) | Potential COC | Noncancer Hazard | | | | | |
|---|---|---|---|---|---|---|---|
| | | Accessible Surface Sediment (a) | Surface Water (a) | Crab Muscle & Hepato | Total Hazard | Percent Contribution to Cumulative Hazard (excluding KM TEQ) (b)(c)(d) | Percent Contribution to Cumulative Hazard (based on KM TEQ) |
| | Pyridine | -- | -- | 2E-01 | 2E-01 | 0.4% | 1% |
| Skin, Blood | Arsenic, inorganic | -- | -- | 4E-01 | 4E-01 | 1% | 1% |
| Urinary | Cadmium | -- | -- | 2E-01 | 2E-01 | 0.5% | 0.5% |
| Thyroid | Cobalt | -- | -- | 1E-01 | 1E-01 | 0.3% | 0.4% |
| GI Tract | Copper | -- | -- | 1E+01 | 1E+01 | 25% | 26% |
| Immune | Mercury | -- | -- | 2E-01 | 2E-01 | 1% | 1% |
| Neurological | Methyl Mercury | -- | -- | 7E-01 | 7E-01 | 2% | 2% |
| | **Total Hazard (excluding KM TEQ) (e)(f)** | **--** | **--** | **5E+01** | **5E+01** | **99%** | **--** |
| | **Total Hazard (based on KM TEQ) (e)(f)** | **--** | **--** | **5E+01** | **5E+01** | **--** | **99%** |

Notes:
Highlighting indicates that the hazard exceeds the goal of protection of a hazard index of one.
(a) Child angler is not assumed to be exposed to sediment or surface water.
(b) Total PCDD/Fs (excluding KM TEQ)—contains contribution of all PCDD/F congeners.
(c) Total DL-PCBs (excluding KM TEQ)—contains contribution of all DL-PCB congeners.
(d) Individual congener percentage contributions were not included in total percent cumulative hazard.
(e) Includes hazard posed by other potential COCs not shown in table.
(f) The sum of percent contribution may not be the same as when the individual values are summed due to rounding.

The cumulative potential cancer risks for the CTE scenario for a crab muscle and hepatopancreas diet are within the NCP risk range. For noncancer HIs, the only CTE target organ-specific HI greater than 1 (USEPA 1991d) is for reproductive effects (DLCs), where the HI is 4, regardless of TEQ approach.

*8.1.4.3 Direct Contact with Sediment and Surface Water*

Cumulative potential cancer risks and noncancer HIs associated with direct contact with accessible surface sediment and surface water in the NBSA while angling, swimming, wading, or boating are within or below the NCP risk range of $10^{-6}$ to $10^{-4}$ and below the noncancer protection goal of an HI of 1 (USEPA 1991d).

*8.1.4.4 Identification of Potential Chemicals of Concern*

Potential COCs were identified in cases when the potential cumulative cancer risk or noncancer HI for a receptor exceed $10^{-4}$ or 1, respectively. In these cases, potential COCs were any COPC with an individual pathway cancer risk greater than $10^{-6}$ or noncancer HI greater than 0.1. The following table summarizes the COPCs for the RME scenario (no COPCs were identified for surface water for either the RME or CTE scenario).

| Potential COC | Accessible Surface Sediment | Mixed Fish Diet | Crab Muscle and Hepatopancreas |
|---|---|---|---|
| **Dioxin-like Compounds** | | | |
| 2,3,7,8-TCDD | | X | X |
| 1,2,3,7,8-PeCDD | | X | X |
| 1,2,3,6,7,8-HxCDD | | X | |
| 2,3,7,8-TCDF | | X | X |
| 1,2,3,7,8-PeCDF | | X | X |
| 2,3,4,7,8-PeCDF | | X | X |
| 1,2,3,4,7,8-HxCDF | | X | X |
| 1,2,3,6,7,8-HxCDF | | X | X |
| Total PCDD/Fs (excluding KM TEQ) | | X | X |
| Total PCDD/Fs (based on KM TEQ) | | X | X |
| PCB-77 | | X | X |
| PCB-105 | | X | X |
| PCB-118 | | X | X |
| PCB-126 | | X | X |
| PCB-156/157 | | X | X |
| PCB-167 | | X | |
| PCB-169 | | X | X |
| Total DL-PCBs (excluding KM TEQ) | | X | X |
| Total DL-PCBs (based on KM TEQ) | | X | X |
| **Non-DL PCBs** | | | |
| Total Non-DL PCBs | | X | X |
| **PAHs** | | | |
| Benzo(a)pyrene | | X | |
| Dibenz(a,h)anthracene | | X | |
| **Pesticides & Organics** | | | |
| 2,4'-DDD | | X | |
| 4,4'-DDD | | X | X |
| 4,4'-DDE | | X | X |
| Chlordane, alpha (cis) | | X | |
| Dieldrin | | X | X |
| Heptachlor epoxide, cis- | | X | X |
| Heptachlor epoxide, trans- | | | X |
| Nonachlor, trans- | | X | X |
| Pyridine | | X | X |
| **Inorganics** | | | |
| Arsenic, inorganic | X | X | X |
| Cadmium | | | X |
| Cobalt | | X | X |
| Copper | | X | X |
| Mercury | | X | X |
| Methyl Mercury | | X | X |

## 8.2 Conclusions

The conclusions of the BHHRA for the NBSA are summarized below. The results for both the RME and CTE scenarios are discussed; however, risk management decisions are based on the RME scenario.

### 8.2.1 Fish and Crab

Consumption of self-caught fish or crab from the NBSA presents the primary source of potential risk to human health. For the RME scenario, which is intended to represent an upper bound of exposure, the potential cancer risk and noncancer hazards to anglers/sportsman who are assumed to routinely consume their catch (34.6 g/day for an adult and 11.5 g/day for a child for fish, or 21 g/day for an adult and 7 g/day for a child for crab, over a period of 26 years) exceed the NCP risk range of $10^{-6}$ to $10^{-4}$ and a noncancer protection goal of an HI of 1 (USEPA 1991d). The RME cancer risk for the combined adult/child angler sportsman is $8\times10^{-4}$ for both fish and crab consumption, and the noncancer HIs for the child angler are 40 for fish consumption and 50 for crab consumption.

For the CTE scenario, which is based on average exposure levels (3.9 g/day for an adult and 1.3 g/day for a child for fish, or 3 g/day for an adult and 1 g/day for a child for crab over a period of 12 years), the potential cancer risks for the combined adult/child angler/sportsman who consumes fish or crab from the NBSA are within the NCP risk range; however, noncancer HIs for the child angler/sportsman above the noncancer protection goal of 1 (USEPA 1991d) (i.e., 4 for fish consumption and 7 for crab consumption).

The primary potential COCs for fish and crab ingestion are 2,3,7,8-TCDD, PCB-126, and non-DL PCBs, with some pesticides, inorganic arsenic, and/or methyl mercury also contributing to the cumulative risks/hazards for both the RME and CTE scenarios. The percent contribution of key potential COCs for the RME scenario are summarized below.

#### *8.2.1.1 Fish consumption*

- Cancer risk (combined adult/child scenario): 2,3,7,8-TCDD contributes approximately 28% (risk of $2\times10^{-4}$), PCB-126 contributes approximately 31% (risk of $2\times10^{-4}$), and non-DL PCBs contribute approximately 18% or 19%, depending on TEQ approach (risk of $1\times10^{-4}$ regardless of TEQ approach).
  - All PCDD/Fs contribute 33% or 34%, depending on TEQ approach (risk of $3\times10^{-4}$ regardless of approach).
  - All DL-PCBs contribute 36% or 38% for all DL-PCBs, depending on TEQ approach (risk of $3\times10^{-4}$ regardless of approach).
  - Minor contributors include pesticides (approximately 5%, maximum risk among pesticides of $2\times10^{-5}$ for dieldrin) and inorganic arsenic (approximately 4%, which equates to a risk of $3\times10^{-5}$).
- Noncancer hazard (child scenario): 2,3,7,8-TCDD contributes approximately 19% (HQ of 8), PCB-126 contributes approximately 20% (HQ of 9), and non-DL PCBs contribute approximately 33% or 32%, depending on TEQ approach (HI of 10, regardless of approach).
  - All PCDD/Fs contribute 22% or 23%, depending on TEQ approach (HI of 10, regardless of approach).
  - All DL-PCBs contribute 24% or 26%, depending on TEQ approach (HI of 10, regardless of approach).

- Minor contributors include pesticides (approximately 8% or 9%, depending on TEQ method, maximum HQ among pesticides of 2 for 4,4'-DDD) and methyl mercury (approximately 6%, which equates to an HQ of 2).
- Target-organ-specific HIs greater than 1 include reproductive (DLCs), whole-body (non-DL PCBs), liver (pesticides, organics), and neurological (methyl mercury).

*8.2.1.2 Crab consumption*

- Cancer risk (combined adult/child scenario): 2,3,7,8-TCDD contributes approximately 52% (risk of $4\times10^{-4}$), PCB-126 contributes approximately 19% (risk of $2\times10^{-4}$), and non-DL PCBs contribute approximately 8%, regardless of TEQ approach (risk of $7\times10^{-5}$).
  - All PCDD/Fs contribute 59% or 60%, depending on TEQ approach (risk of $5\times10^{-4}$ regardless of approach).
  - All DL-PCBs contribute 23% or 24% for all DL-PCBs, depending on TEQ approach (risk of $2\times10^{-4}$ regardless of approach).
  - Minor contributors include inorganic arsenic (approximately 6%, which equates to a risk of $5\times10^{-5}$) and pesticides (approximately 2%, maximum risk among pesticides of $1\times10^{-5}$ for dieldrin).
- Noncancer hazard (child scenario): 2,3,7,8-TCDD contributes approximately 33% (HQ of 20), copper contributes approximately 25% to 26%, depending on TEQ approach (HQ of 10), PCB-126 contributes approximately 12% (HQ of 6), and non-DL PCBs contribute approximately 14%, regardless of TEQ approach (HQ of 7).
  - All PCDD/Fs contribute 38%, regardless of approach (HI of 20).[8]
  - All DL-PCBs contribute 15%, regardless of TEQ approach (HI of 7).
  - Minor contributors include pesticides (approximately 3%, regardless of TEQ method, maximum HQ among pesticides of 0.5 for 4,4'-DDD) and methyl mercury (approximately 2%, which equates to an HQ of 0.7).
  - Target-organ-specific HIs greater than 1 include GI tract (copper), reproductive (DLCs) and whole-body (non-DL PCBs).

As discussed in Section 7.3.3, the TEFs for DL compounds carry considerable uncertainty, particularly for some of the DL-PCBs. Consistent with USEPA (2010a), a sensitivity analysis was conducted to illustrate the impact of the TEFs on the overall risk estimates and percent contribution of individual congeners or groups of congeners. For all congeners except 2,3,7,8-TCDD, the lower- and upper-bound TEFs were the 10$^{th}$ and 90$^{th}$ percentiles from *in vitro* and *in vivo* studies included in the relative effects potency (ReP) database (USEPA 2010a). The TEF for 2,3,7,8-TCDD remains constant in all scenarios. Accordingly, while the estimated risk for 2,3,7,8-TCDD remains constant, the contribution to risk can change, as can the relative contributions of all PCDD/Fs, all DL-PCBs, and all PCBs (non-DL and DL-PCBs). For example, for the

[8] The HQ for 2,3,7,8-TCDD is also shown as 20 due to rounding; however, other PCDD/F congeners also contribute to the total HI for all PCDD/Fs.

combined adult/child angler/sportsman who consumes a mixed fish diet, the percent contribution for 2,3,7.8-TCDD increases from 28% to 44% when using the lower-bound TEFs, but decreases to only 1% when using the upper-bound TEFs. Conversely, the percent contribution to overall risk for Total PCBs (DL-PCBs and Non-DL PCBs) increases from 37% when using lower-bound TEFs to 98% when upper-bound TEFs are used. Similarly, for crab muscle and hepatopancreas consumption, the percent contribution of 2,3,7,8-TCDD increases from 52% to 70% when using the lower-bound TEFs but decreases to approximately 2% when using the upper-bound TEFs. The percent contribution to overall risk for Total PCBs (DL-PCBs and Non-DL PCBs) increases from 16% when using lower-bound TEFs to 96% when upper-bound TEFs are used (see Section 7.3.3).

The specific species or tissue type(s) that make up a fish or crab diet can influence the estimated risk, because some species or tissue types have been shown to have higher tissue burdens of bioaccumulative chemicals than others. Fillet data were collected for five fish species from the NBSA: American eel, bluefish, striped bass, summer flounder, and white perch. The estimated cancer risks associated with consumption of any combination of these fish species exceed the NCP risk range for the RME scenario, but not the CTE scenario. The estimated noncancer HIs exceed the noncancer protection goal of an HI of 1 (USEPA 1991d) for both the RME and CTE scenarios. The estimated cancer risks associated with consumption of crab muscle only are approximately a factor of 5 to 6 lower than for consumption of crab muscle and hepatopancreas combined, but remain above the NCP risk range for the RME scenario. For noncancer effects, the noncancer HIs for a muscle-only diet are also approximately a factor of 3 lower than for muscle and hepatopancreas combined, but remain above the noncancer goal even for the CTE scenario. The risks/hazards to consumers of the crab hepatopancreas only would be much higher than the risks for consumers of muscle only or combined muscle and hepatopancreas.

### 8.3 Sediment and Surface Water

The cumulative potential cancer risks and noncancer HIs associated with direct contact with accessible surface sediment and surface water in the NBSA while angling, swimming, wading or boating, are much lower than those associated with fish or crab consumption and are within or below the NCP risk range of $10^{-6}$ to $10^{-4}$ and below the noncancer protection goal of an HI of 1 (USEPA 1991d).

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